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A TREATISE  
ON  
THE DISEASES  
OF THE  
NERVOUS SYSTEM.

BY

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*ILLUSTRATED WITH LITHOGRAPHS, PHOTOGRAPHS, AND  
TWO HUNDRED AND EIGHTY WOODCUTS.*

VOLUME I.



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## PREFACE.

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IN the following pages I have endeavoured to give a tolerably full account of the diseases to which the nervous system is liable, with the exception of the psychoses, which form a sufficiently large and important group to demand a separate treatise. It would be invidious to single out for special mention a few of the many authors whose works I have had occasion to consult; I must, therefore, ask them all to accept the simple acknowledgment conveyed in the insertion of their names in the text.

A large number of the illustrations are borrowed from Henle's "Anatomie," Landois' "Physiologie," Heath's "Anatomy," Tibbits' "Medical Electricity," Walton "On Diseases of the Eye," and Gamgee's translation of Hermann's "Physiology," as well as from the writings of Flechsig, Duchenne, Charcot, Erb, and several other authors. A considerable number, however, are original, most of these being drawings taken by Dr. A. H. Young from my own sections.

I have to express my obligations to several of my colleagues at the Manchester Royal Infirmary for the kindness and courtesy with which they have placed cases at my disposal, and for much valuable aid during the progress of the work through the press.

In common with all other medical workers in Manchester, I owe to Mr. T. Windsor a debt of gratitude which can never be

repaid; inasmuch as, with but a small annual sum at his disposal for the purchase of books, he has, with singular energy and life-long devotion, placed within reach of the profession one of the best medical libraries of reference in the kingdom.

I have already mentioned the name of Dr. A. H. Young, Pathological Registrar to the Manchester Royal Infirmary; my thanks are due to him in a special manner not only for his excellent drawings, but also for much valuable assistance in preparing the anatomical part of the work, and the sections on shock and concussion.

I am indebted to Dr. Lindemann for preparing a copious index, upon which he has expended great care and labour.

My chief thanks, however, are due to Dr. Steell, Resident Medical Officer of the Manchester Royal Infirmary, and I do not know how to express them adequately without appearing to involve him in a joint responsibility not only for what there is of merit in the work but for its many shortcomings also. He has carefully assisted me in correcting all the proof-sheets, and has in doing this subjected the entire text to the wholesome ordeal of a severe criticism, whereby the work has gained largely both in accuracy of statement and conciseness of style.

It was my intention to have supplied a copious bibliography, but the unexpected size which the work has attained compelled me to abandon this idea. I have had to content myself with giving a few general instructions to the reader, which will, I trust, enable him to find without much difficulty the works and papers to which reference is made in the text.

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## **BOOK I.**

### **GENERAL PATHOLOGY OF THE NERVOUS SYSTEM.**





# GENERAL PATHOLOGY OF THE NERVOUS SYSTEM.

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## CHAPTER I.

### INTRODUCTION—THE STRUCTURE AND FUNCTIONS OF THE NERVOUS SYSTEM.

BEFORE entering upon the consideration of the nervous system, I may be permitted to make a few remarks, by way of introduction, on the fundamental properties of living matter. Every kind of living matter is found to be a complex combination of carbon, hydrogen, oxygen, and nitrogen, and some secondary, but still essential, constituents. The peculiarities of the substance of living bodies, therefore, do not depend on the existence of a new kind of matter, but on new combinations or collocations of constituents with which we are familiar in their separate states, and in their simpler combinations.

But a great many persons cannot bring themselves to believe that the forces which produce the forms and actions of living beings are the same as those which are operative in the inorganic world. Everyone admits that certain kinds of living action, such as the motions of the limbs by muscular contraction, are subject to mechanical laws; but some think that the more recondite processes, such as muscular contraction itself, are under the control of a higher principle. An attempt has been made in all ages to formulate this conception. Aristotle assumed the existence of a "vegetative soul" to account for living processes and actions. Van Helmont called the principle of life "Archæus," and Stahl called it "Anima;" while in modern times these ideas have been superseded by the conception of a "vital force" in no way related to the physical forces.

But science, especially in its objective aspect, cannot recognise the dualism which would ascribe some living actions to mechanical forces, and reserve others for the supervision of another force which is in no way related to the former. No living action or process has ever been rescued from the unknown to the known except by the application to it of the laws of inorganic nature. Life does not introduce us to a new order of force, but to new combinations or collocations of the forces which have already become more or less familiar to us in the study of the physical and chemical sciences. In one word, the great law of the conservation of energy is as applicable to the objective study of living beings as to that of any part of the universe within the reach of our observation.

§ 1. *Conservation of Energy.*—The principle of the conservation of energy asserts that force is neither created nor annihilated, and that its quantity in the universe is as unalterable as the quantity of matter. The different forms of force may be transmuted into one another; but nothing is lost in the transfer. Mechanical force may pass into heat; but by suitable arrangements the heat may be reconverted into the original amount of mechanical momentum.

This is not the place to enter on an exposition of the doctrine of the conservation of energy, but I may be allowed to remind the reader that the various forms of force are on ultimate analysis reducible to two kinds—namely, attraction and repulsion. When two bodies which attract each other are separated by a space, the system possesses energy, or a power of performing work. If they are prevented by an external force from obeying their attractions by moving towards one another, the energy of the system is potential; and when all hindrances to their movement towards one another are removed, the energy becomes actual, dynamic, or kinetic. Substances which, instead of attracting, repel each other, must be pressed towards one another by some external force before the system can possess energy; so long as this external force prevents the two repelling bodies from moving away from one another, the energy is potential; and when all hindrances



to the free action of their mutual repulsions are removed, the energy becomes kinetic. The force which removes all the obstacles to the motion of attracting bodies towards each other, or of repelling bodies away from one another, and which thus converts the potential into kinetic energy, is called the liberating or discharging force. Two other terms are employed in describing the transformations of energy, which it is well to remember. When two bodies are so related that a small liberating force renders kinetic their potential energy, then the system is said to be in a position of *unstable equilibrium*, and when the system either possesses no potential energy, or when a relatively large liberating force must be applied before its potential energy can be rendered kinetic, it is said to be in a position of *stable equilibrium*. As a familiar instance of unstable equilibrium, I may take a thin book from my table, and place it on end on the floor. A slight touch at the free end of the book will cause its centre of gravity to project beyond its base, and the earth and the book will then rush towards one another—the slight touch has converted the potential into kinetic energy. Now, however, when the book rests on its side, a relatively large amount of external force must be applied to it before it can give out any actual energy; the earth and the book are in a position of stable equilibrium in relation to one another.

§ 2. *Fundamental properties of living matter.*—Let us now proceed to the consideration of the fundamental properties of living matter. The most casual observation must convince anyone that organisms are not an aggregate of materials heaped up in any fashion; but that, however diverse may be their forms, they are constructed in an orderly manner. Now, suppose that an intelligent inhabitant of Central Africa, on visiting this country, had been struck with the utility of brick buildings, and wished to acquire a knowledge of their mode of construction, what would be the best means of imparting the desired information? We should show him that the house is mainly composed of walls; that each wall, and each part of a wall, is made up of bricks. Having shown that the unit of composition, or, in other words, the structural unit of the

house, is a single brick, we should then direct him to make a special study of the mode of formation and properties of the brick ; and after he had mastered the details of brickmaking, he could then be shown how brick is united to brick to form a wall, and so on, until he was taught how a house is built. In short, in order usefully to study such a simple construction as a brick house, there must first be either a real or an ideal breaking down of the building into its constituents, and then either a real or an ideal putting together of the constituents to form the building. Analysis and synthesis are both necessary, but the former ought to precede the latter.

If we subject organisms, in the first place, to ideal analysis, we shall find that each is composed of certain mechanisms or organs ; and that each organ is composed of certain definite arrangements of tissues ; and, finally, that each tissue is composed of structural units, which, for the sake of illustration, we may call 'organic bricks.' To make a real analysis of a living organism is a much more difficult matter than to make a similar analysis of a brick building, since the living properties of the structural unit which we wish to study generally escape us in the act of making the analysis. But, fortunately, even in the higher organisms there are some units—such as the white blood-corpuscles—which can be kept alive for some time after detachment from the parent organism, and in which the fundamental properties of the structural unit may be usefully studied. We generally, however, avail ourselves of the analysis which nature has made ready to our hands. It is not possible to build a house with a single clay brick ; but it is possible for a single organic brick to constitute a complete living individual. The lowest living organisms—if indeed they are entitled to be called organisms, since they do not possess real organisation—are mere specks of albuminoid matter, the protoplasm of biologists, and correspond to the structural units of the higher organisms. It is by the study of these lowly organisms that we must expect to ascertain the fundamental properties of living matter.

Amongst these primordial organisms, probably the best adapted for the study of the fundamental properties of life are the amœbæ, both on account of their simplicity and of their

similarity to the white corpuscles of vertebrate blood. There are several kinds of these, but all of them may be arranged under three leading orders:—

- (1) Organisms consisting of a simple speck of protoplasm, such as the *Protamœba primitiva* found by Hæckel in fresh water.
- (2) Organisms more or less similar to the above, but possessing a distinct nucleus.
- (3) Similar organisms, exhibiting, along with the granular protoplasmic interior or endosarc, a more solid external layer or ectosarc. The external layer, instead of being a membrane, is sometimes represented by a shell; but the former, owing to its similarity to the membrane surrounding the cells which form the tissues of the higher organisms, is of more importance to us.

This classification shows that the protoplasm is the fundamental substance, without which no organism can exist, and that the other constituents found in the unit are mere specialisations of this fundamental substance. A knowledge of the primary properties of life will, therefore, be best gained by the study of the first order of unit.

§ 3. *Protoplasm*.—If one of these lowly organisms be watched, it is seen to throw out processes of its substance, and then to retract them—changes which are accompanied by a flux and reflux of its granular substance,—and it is able by this means to perform a certain amount of locomotion. The amœba is *contractile*.

Amœboid movements occur under two conditions. In the first place, contact with foreign bodies, and chemical and electrical agents, call forth these movements. These disturbing causes act as liberating forces in rendering kinetic the energy potential in the protoplasm; and, as is usual in such cases, the energy set free is out of all proportion to the cause which determines the transformation. A disturbing force which determines a discharge of energy in living matter is called a *stimulus*. In the amœba, the application of a stimulus leads to movement, but in some cases the energy set free by the discharge assumes



the form, not of contraction, but of heat. We want, therefore, a generic term to express the fact that when living matter is acted on by a stimulus there is an active development of energy, whatever the form the energy assumes. The term employed for this purpose is *irritability*, and a tissue which responds to a stimulus by an expenditure of energy is said to be *irritable*. Irritability, therefore, is the genus, of which contractility is the most important species; the former is, but the latter is not, co-extensive with life.

But in the second place, the movements of the amœba cannot always be referred to the action of external stimuli; the energy is frequently set free in consequence of internal changes, and the movements are then called *spontaneous* or *automatic*. It may therefore be said that protoplasm is *irritable* and *automatic*.

We have already seen that when there is an expenditure of energy, matter has fallen from a relatively unstable to a relatively stable position, in which it possesses a comparatively small amount of energy or none. And if no counterbalancing actions were proceeding, the store of energy in the substance of the amœba would soon be exhausted; and, indeed, this store may be exhausted at one stroke by the application of a powerful stimulus. This action may be very readily watched with an organism closely allied to the amœba—the *Protococcus pluvialis*. If, while watching its movements under the microscope, a drop of dilute alcohol, or a weak solution of quinine or strychnine, be placed at the edge of the cover-glass, when the chemical agent makes its way to the organism its ordinary movements give place to one or two violent contractions, which are followed by quiescence. In this condition no further stimulation will evoke movements; the protoplasm has lost its irritability. There is one violent explosion of energy, ending in complete expenditure of the stock. In this case the expenditure is followed by permanent loss of irritability, or death, probably because the chemical stimulus continues to act upon it; but when the action of the stimulus is temporary, as with an electric stimulus, the loss of irritability induced may be followed by gradual restoration. But, whether the loss of irritability be temporary or permanent, when it is caused by the sudden appli-

cation of a violent stimulus the effect is called *shock*. The loss of irritability may also be caused by the prolonged application of a moderate stimulus, if the conditions necessary to the acquirement of a new stock of irritable matter are prevented. Under these circumstances the state is called *exhaustion*.

The allusion just made to the absorption by the protoplasm of the amœba of a new stock of energy leads us to the consideration of a second group of fundamental properties of living matter. Certain substances serving as food, when received into its body, are manufactured into new protoplasm. This process is called *assimilation*. The protoplasm, however, is continually undergoing chemical change (metabolism); the old protoplasm is broken up, and the products of disintegration are cast out of the body as *excretions*. This process is termed *disassimilation*. Some of the products, however, are probably retained within the body for a time, and used in the solution and preliminary changes of the raw food, and these are termed *secretions*.

But all the protoplasm formed by an amœba is not immediately disintegrated; some of it is added to its substance, constituting *growth*.

§ 4. *Nucleus*.—When the amœba attains a certain size, it generally resolves itself by fission, or by other means, into two or more parts, each of which is capable of living as a new individual, which passes through phases of life similar to those of the parent from which it is derived. This process, then represents the first beginnings of two great living functions—reproduction and inheritance. So far we have spoken of the protoplasm as being the fundamental agent which exhibits all the properties of life, and there can be little doubt that it exhibits the great functions at present under consideration. When, however, the protoplasm becomes so far differentiated as to possess a nucleus, the latter constituent appears to preside in a peculiar manner over the functions of reproduction and inheritance. In the second order of amœbæ the nucleus is seen to divide into two or more parts prior to the fission of the protoplasm. When these units, instead of parting company and each leading a separate life of its own, aggregate so as to form a compound organism or a compound tissue, this process of fission subserves



the great function of growth, since the organism or tissue grows not so much by increase in the size, as by multiplication in the number of units.

§ 5. *Cell-Membrane*.—Two of the constituents of the unit have been passed in review. The nucleus, being of denser consistence than the protoplasm, and presiding, as we have supposed it to do, over the great functions of reproduction and inheritance, tends to make the offspring like the parents. The protoplasm, on the other hand, being of a semi-fluid consistence, and adapting itself, as it does, to different circumstances by changes of form, tends to introduce variety, and to make the progeny unlike the parents. But this power of adaptation, although very varied in degree, is only of one kind—namely, contraction of the protoplasm; and the fact that a stimulus produces a contraction at one time does not enable the protoplasm to respond better to a similar stimulus a second time. So far there is no principle of improvement or progress. Under these circumstances we turn to the third constituent of the unit, namely, the cell-membrane. The membrane in the first instance limits to a large extent the degrees of adaptation of the protoplasm. Being of denser consistence, it offers greater passive resistance to external forces, and the flow of nourishment towards the interior is retarded, so that the amount of irritable matter at the disposal of the organism is diminished. The powers of the organism are thus greatly limited by the membrane. But if the membrane diminishes the degrees, it increases the kinds of adaptation. The increased density of the ectosarc enables it to offer a certain amount of passive resistance to external forces, and the endosarc is therefore more free to expend its energies in internal action. And although the membrane is not an active agent in producing adaptations, it tends to fix and perpetuate those adaptations which are frequently repeated, and in the compound organisms thus makes possible progressive improvement through successive modifications.

§ 6. *Cell-Contents*.—Besides the protoplasm and nucleus,

other substances are found enclosed in the cell-membrane, technically called cell-contents. Sometimes the cell-contents are fluid, and then generally belong to the secretions or excretions already mentioned. Generally, however, they are solid, such as inorganic crystals, organic concretions like starch corpuscles, fat granules, chlorophyl, hæmoglobin, and various pigmentary granules. Some of these, as starch and fat, are stores of potential energy; chlorophyl and hæmoglobin appear to be subservient to the respiratory function, while the accumulation of pigment has obscure relations to other special functions.

§ 7. *Assimilation and Disassimilation.*—The two most fundamental processes of life, then, are assimilation, or the process by which irritable matter is formed and energy rendered potential; and disassimilation, or that by which the irritable matter is broken up and energy rendered kinetic. These processes underlie and render possible the other processes of life. When assimilation is in excess of disassimilation—or, in more general terms, when integration is in excess of disintegration—growth takes place; and when growth proceeds to a certain extent, a portion of the material is given away for the production of a new individual. On the other hand, the energy rendered active during the disintegration of the irritable matter is chiefly applied in those lower organisms to the execution of movements, and, to some small extent, to the production of heat. All these functions, then, aid each other in the preservation of the individual and of the race. Were that addition to the bulk of the organism which constitutes growth to cease, reproduction would soon become impossible, since, in the absence of the former, the process of fission, which is the essential factor of the latter, would soon diminish the size of the organism to a point incompatible with life; and that growth could not proceed far without reproduction is too manifest to require pointing out. These functions are in their turn dependent on the contractile power of the protoplasm, since without the latter property the circulation of materials which is necessary to life would cease.



§ 8. *Antagonism between Growth, Reproduction, and Action.*—But although the growth, reproduction, and motor functions of these simple organisms aid each other in the maintenance of the organism, there are also fundamental antagonisms between them. Whenever multiplication occurs, it is clear that the parent individual must part with a certain amount of material, and that its bulk must be diminished by the bulk of the matter given away. Nutriment may be applied either to the growth of the parent or to the production of one or more new individuals; but it cannot at one and the same time be applied to both purposes. Every bit of material given away to form a new unit is a deduction from the size of the parent; and when the latter frequently parts with material for the production of progeny, its size is diminished in a corresponding degree; in other words, rapid reproduction is accompanied by units of small size. Again, an organism may use its nutriment in executing movements; and when movements are executed, the nutriment must be transformed, just as coal must be transformed in order to put our locomotives in motion; and when it is transformed, it can neither be added to the size of the parent, nor devoted to the production of a new individual. A fresh supply of coal may be disposed of in several ways: it may be added to the previous stock, given away, or burnt; but it cannot at one and the same time be stocked, given away, and burnt. And so it is with respect to the disposal of nourishment by protoplasm: it can be applied to increase the size of the organism, to its multiplication, or to the execution of movements; but so much of it as is applied in one direction cannot be applied in either of the other directions.

Once more: when the protoplasm is surrounded by a dense membrane, the flow of nourishment into it is much retarded; and as an organism can neither expend, nor add to its bulk, nor give away what it does not receive, one surrounded by a dense membrane (or shell) can neither move actively, nor increase rapidly in size, nor multiply quickly. The membrane gives form, and fixity, and permanence, and resisting power to the protoplasm; but these properties are gained at the expense of the motor functions, growth, and reproduction. There is, in



short, a mutual antagonism between each and all of these functions, so that increase of activity in one direction involves decrease of activity in the other directions.

In the construction of the higher animals, the units, instead of parting company, and each living an independent existence, aggregate, and every new unit formed becomes incorporated with the general mass. But a simple aggregation of living units having similar properties would not confer any advantages on the organism; while the units themselves would greatly lose by the fact of their contact. One effect of the contact is that the surfaces exposed to the environment, and through which food can be absorbed, are greatly diminished, and their opportunities of obtaining food when associated are less than when each unit is free. When, therefore, we see an organism of considerable dimensions, we may be quite sure that the units have, by the very fact of their association, gained advantages in certain directions, even if these are counter-balanced by losses in other directions.

§ 9. *Antagonism between the Size of Units and Absorption of Nourishment.*—It has just been stated that both the presence of a dense cell-membrane, and the association of units to form an aggregate, must retard the exchanges of material that are constantly taking place between the substance of living cells and the surrounding substances which are utilised by them as food; and it may now be remarked that simple increase in the size of the units must necessarily have a similar effect. That an increase in the size of a cell is followed by a relative diminution of material exchanges is readily proved by the fact that the surface which a large cell presents for the absorption of nourishment does not increase in a degree proportionate with its bulk. The mass of a body increases as the cube, while the surface only increases as a square, of the dimensions. When, for instance, a cell has doubled its dimensions its mass is eight times, while its surface is only four times, the original size. It is evident, therefore, that a small cell presents, in proportion to its bulk, a larger surface to its environment for the absorption of nourishment than a large cell, and consequently material exchanges take place more readily in the former than in the latter. It is

not very easy to find inductive evidence illustrative of this law, inasmuch as increase in the size of the cell is usually associated with increase in the density of its cell wall; so that it is not possible to determine how much of the diminution of the material exchanges is to be attributed to each factor. The white blood-corpuscles are of small size, and they are placed in the most favourable circumstances for obtaining food, inasmuch as their surfaces are bathed in a highly nutrient fluid; while large cartilage cells are, on the other hand, always placed in the most unfavourable circumstances for obtaining nourishment, inasmuch as they are removed to a considerable distance from the vessels by which they are supplied with nutrient fluid. But during health the supply of nourishment to the tissues must be proportionate to the demand; hence it may be inferred that rapid material exchanges take place in the former, and that the exchanges in the latter are very slow. It is impossible, however, to determine how much of the difference in the nutritive activities of the two units is to be attributed to the difference in their sizes, and how much to the absence of a cell wall in the case of the white corpuscles and the presence of a dense inter-cellular substance in the case of cartilage cells. It is worthy of remark, however, that in articular cartilage the smaller and embryonic cells are found nearer the blood-vessels than the larger and older cells; and there can be little doubt that a similar relationship exists between the blood-vessels and embryonic cells in other tissues.

§ 10. *Differentiation of Structure and Specialisation of Function.*—In social organisms growth is rendered possible by the specialisation of function which is denominated "division of labour;" and the formation of a society of living units renders possible a similar specialisation of function which has been aptly named "a physiological division of labour." This "division of labour" does not introduce us to a new property of protoplasm, but merely to a new principle, whereby the properties with which we are already familiar may be combined in various ways. Certain groups of the constituent units become adapted for the manifestation of one or a few only of the fundamental properties of protoplasm, to the complete sub-



ordination of the other properties. This can be done with advantage to the organism only on condition that other groups of units become adapted for the manifestation of the properties which have become subordinate in the first group. In the higher organisms one group of units becomes pre-eminently contractile; a second pre-eminently irritable and automatic; other groups become respectively secretory, excretory, respiratory, and metabolic; while another group becomes specially adapted for reproduction; and a final group possesses only a passive or mechanical function.

"The physiological division of labour" has for its morphological correlative "differentiation of structure;" and the groups of units which assume special functions, correspond to the various tissues. In the formation of the tissues the cell-membrane assumes great importance. It is evident that if the units of the tissues of a compound organism were entirely composed of the semi-fluid substance termed protoplasm, they would not have sufficient tenacity to stick together. In the formation of structure, therefore, the units must be surrounded by a membrane; and when the membranes of adjoining units become amalgamated, they are called *intercellular substance*.

The disposition of the membrane or of the intercellular substance, and the relative amounts of the different constituents of the unit, must vary according to the function of the tissue. When the function of a tissue is of a passive nature—such as that of cartilage—a relatively large amount of intercellular substance is present; and if, in addition, the tissue requires rigidity, the intercellular substance is strengthened by the deposition of other materials, such as carbonate and phosphate of lime in bone. When, on the other hand, the tissue is actively growing, the proportion of intercellular substance to protoplasm is small, as in granulation tissue; and when the units multiply rapidly, the intercellular substance disappears, the nucleus becomes conspicuous, and the tissue breaks up into separate units, as in pus. We also meet with independent units, without membrane, in the white corpuscles of the blood, whose functions appear to be of a metabolic, and therefore of an elementary, character. When material is stored up in the tissue for future use, then the cell-contents come into prominence. The tissues

which perform active functions, as muscle and nerve, must be composed of a due admixture of membrane and protoplasm. The transformation of the protoplasm supplies the motor force; and the disposition of the membrane gives fixity to the arrangements, and determines the direction in which the energy is expended. The burning of coal in our furnaces supplies the power which moves our factories; but the structural arrangements of the machinery determine whether the factory shall be adapted for weaving or spinning, or for the manufacture of wool, cotton, or silk. And as the structural arrangements determine the functions of the factory, so it is with living tissues. The active tissues must therefore have a much more elaborate arrangement of the intercellular substance than the passive tissues, so that the energy given out by the transformation of their protoplasm may be directed to definite ends; and while the intercellular substance must be sufficiently dense to give fixity to the arrangements, it must not be so dense as greatly to retard the flow of nourishment from the blood to the protoplasm: otherwise the function of the tissue would be diminished as surely as a scanty supply of coal would diminish the work of the factory. It will be readily understood, from the antagonism between reproduction and active expenditure of energy, that a tissue which is performing an active function cannot have its units multiplying rapidly; and if from any cause these units begin to multiply, the capacity of the tissue for the display of function will become impaired.

§ 11. *Integration of Structure.*—In the formation of the higher organisms, the process of differentiation which ends in the formation of the tissues must be accompanied at every step by corresponding integration, whereby the tissues become united to form mechanisms or organs. Each organ is built up of a combination of tissues, and this is especially true of those organs which perform active functions. The fundamental tissue of each organ corresponds to its main function; but it is packed together to form an organ by means of a passive tissue.

Simultaneously with the integration of the tissues to form organs, there goes on a corresponding integration of organs to form the individual. Some of these organs are devoted to the



accumulation and elaboration of nutriment; others to its absorption and distribution; and others to the active expenditure of the nourishment, as in animal locomotion,—and this necessitates the formation of other arrangements for the removal of waste materials; and lastly, the integration is completed by the various tissues and organs being brought into close connexion under a central regulative organ, by means of which the various actions of the individual are duly co-ordinated.

§ 12. *Passage from the general to the special in both structure and function.*—We have seen that, when an organism consists of an aggregation of units without any definite arrangements, each part is able to perform all the vital functions. Each part is irritable, automatic, contractile, metabolic, excretory and reproductive; and it is only as fast as this originally uniform tissue becomes differentiated that each part acquires the power of performing more perfectly a few functions, and ultimately one special function, while losing to a greater or less extent the power of performing the general functions. The expanded tentacle of a zoophyte, on being touched, immediately contracts, and after a time it expands, apparently from its own inherent activity. The same tissue is sensitive to external impressions or irritable, as well as automatic and contractile. Of essentially the same nature appears to be the mechanism concerned in the movements of the leaves of carnivorous plants: the tissue which receives the impression also closes the leaves by its contraction, and after a time expands them by its own inherent activity. Such actions foreshadow the functions of nervous tissues, but they fall short of true nervous action. In *Hydra* the internal end of an ectodermic cell is prolonged into a process, which assumes the contractile function; while the external end of the cell becomes specially sensitive to external impressions. The internal end being shielded from external influences, tends to contract only when it receives a stimulus through the external end; and the latter always tends to lose its contractile property, the more its exposed position requires it to determine when the former shall be made to contract. In other words, the internal

end performs the work, while the external determines when the work shall be done: the one is *operative*, the other *regulative*.

This differentiation of structure is carried still further in *Beroë*, where the internal and external ends of the ectodermic cell are represented by two different cells connected by a thin fibre. The changes set up in the external or sensitive cell are conducted through the fibre to the internal cell, which it excites to contract. This new arrangement of fibre introduces us to a new special function. A part of the tissue is set apart for conveying waves of disturbance from the sensitive cell to the work cell. By this means a molecular motion on the surface is followed by a molar motion in the interior, the two being at some distance from each other, and co-ordinated by the internuncial function of the fibre. The next step of development consists in the differentiation of the external or sensitive cell into two cells; the one of which becomes specialised for responding to external stimulation alone, and the other for automatic action. The latter will perform its functions best by being shielded from external influences, and it will therefore be withdrawn from the surface of the body; while the former will retain its superficial position in order that it may respond the better to external changes; and each of these, by being relieved of one kind of action, will perform more efficiently the action or actions it retains. The fibre which originally connected the ectodermic cell and the contractile process now connects the latter with the automatic cell; and a new fibre is required to connect the automatic with the sensitive cell. The automatic cell is a centre to which disturbances originating in the sensory cell are conveyed, and from which issue impulses to the work-organs; hence the fibre which connects it with the sensory cell is rightly called the *afferent*, and that uniting it with the work-organs the *efferent* fibre. The automatic cell is at present represented as being engaged, both in spontaneous action and in modifying afferent impulses previous to their being passed on to the efferent fibres. But the central cell becomes by-and-by differentiated into two cells, the one of which is restricted mainly to automatic action, and the other to the co-ordination of afferent impulses previous to



their conversion into the outgoing discharge. The latter constitutes what is called *reflex action*.

The fundamental fact, however, which concerns us at present, is that the active elements of which the nervous tissues are composed consist of *cells* and *fibres*. We must now glance rapidly at the more important properties of these cells and fibres.

§ 13. *Ganglion-Cells*.—The ganglion-cells possess granular contents, and a vesicular nucleus with a nucleolus. They vary much in size and form.

(1) *Apolar Ganglion-Cells*.—Some of the cells are small, generally spherical or ovoid, and have a regular uninterrupted outline. These cells are also called apolar, from the fact that they do not possess any processes. It is probable that the majority of such cells are embryonic forms.

(2) *Caudate Ganglion-Cells*.—Other ganglion-cells are much larger than those just described. They possess a definite cell-wall, and have one, two, or more long processes issuing from them. These cells are called stellate, or caudate ganglion-cells (*Fig. 1, I*), according to their form, and unipolar, bipolar, or multipolar according to the number of their processes. Each cell possesses a large oval nucleus, situated near its centre. The nucleus is composed of a well-defined membrane and an intranuclear network, and its centre is occupied by a highly refractive nucleolus. The body of the cell is composed of numerous minute fibrils, which are connected with each other in a network.

(3) *Processes*.—The processes of the ganglion-cells are of two kinds—branched (*Fig. 1, I y*) and unbranched (*Fig. 1, I z*). The former are, like the body of the ganglion-cells, composed of fibrils which run in a longitudinal direction, and pass in a fan-like manner from the processes into the body of the cell. These processes divide and subdivide dichotomously, so as to form a network of fine filaments (*Fig. 1, I y*). The unbranched processes are pale and finely striated bands, which represent and are continuous with the axis-cylinder of the medullated nerve-fibres. The unbranched or axis-cylinder process is usually single, although occasionally two of these processes are attached to one cell. Most ganglion-cells, and especially the larger ones, are surrounded by a pericellular, or lymph-space, through which

FIG. 1.

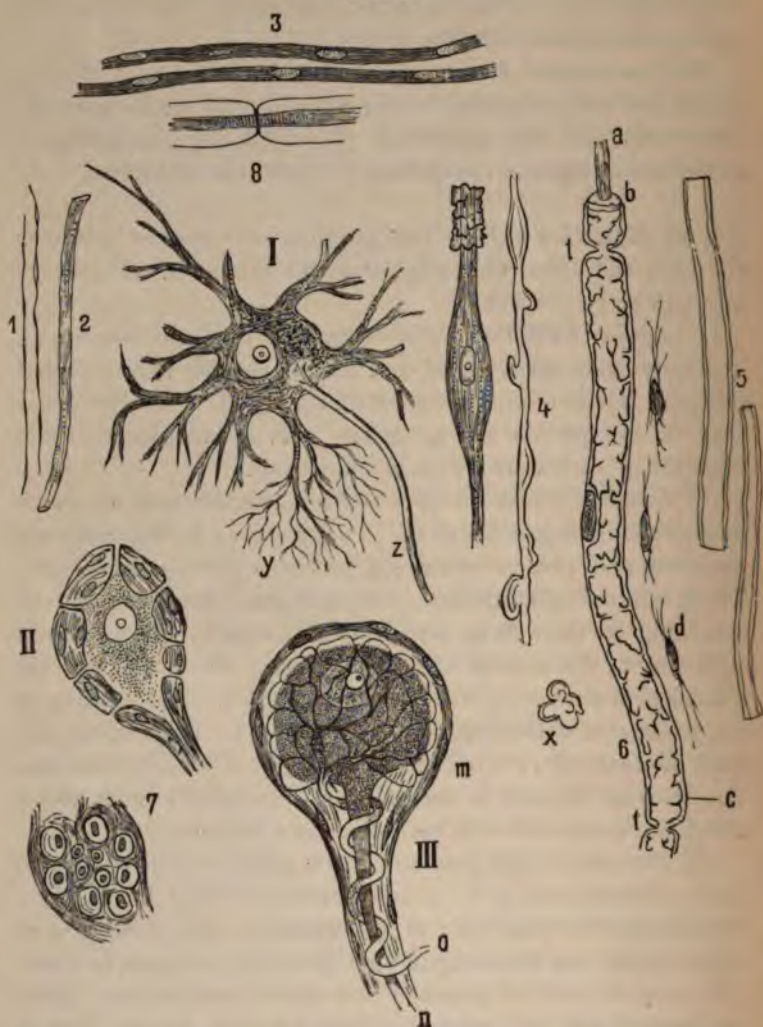


FIG. 1. (From Landois' "Physiologie.")—1, Primitive Fibrillae. 2, Axis Cylinder. 3, Remak's Fibres. 4, Medullated Varicose Fibres. 5, 6, Medullated Fibres, with the Sheath of Schwann; c, Neurilemma; t, t, Ranvier's Nodes; b, Medulla; d, Cells of the Endoneurium; a, the Axis Cylinder; x, Drop of Myeline. 7, Transverse section of Nerve Fibres, with distinct Axis Cylinder, Medullary Sheath, and Perineurium; 8, Nerve Fibres treated with nitrate of silver, presenting the appearance of a cross at the Node. I, Multipolar Ganglion Cell of the Spinal Cord; z, Axis Cylinder process; y, Protoplasmic processes. On the right of I a Bipolar Ganglion Cell is represented. II, Peripheral Ganglion Cell, surrounded by a Capsule lined by Endothelial Cells. III, Ganglion Cell, with Spirally-twisted Fibre; m, Capsule; n, Axis Cylinder process; o, Spirally-twisted Fibre.



the processes of the cell pass. The capsule in which the ganglion-cell is enclosed is formed of a hyaline membrane similar to and indeed continuous with the sheath of Schwann of the nerve-fibre. This capsule is lined by a layer of small, more or less polyhedral or flattened protoplasmic cells, each containing a round or slightly oval nucleus, and forming at times an almost complete epithelioid lining (*Fig. 1, II*). The number of these lining corpuscles varies in different ganglia, and they are most numerous near the axis-cylinder. The hyaline membrane of the capsule is continued around the unbranched process of the cell, while the corpuscles lining the capsular membrane also appear at intervals as nuclei surrounded by a thin layer of protoplasm interposed between the axis-cylinder and the sheath of Schwann, and between the latter and the medullary sheath when once the fibre has become medullated. In the frog the bipolar ganglion-cells of the abdominal part of the sympathetic system are very peculiar, inasmuch as one process becomes twisted in a corkscrew manner round another process, the former being called the spiral (*Fig. 1, III o*) and the latter the straight fibre (*Fig. 1, III n*). These cells were discovered by Beale, and are consequently named after him. The spiral is generally thinner than the straight fibre, and there is always an accumulation of small nuclei where it leaves the cell substance. The spiral fibre is at first thin, but soon becomes thicker and transformed into a medullated nerve-fibre; but the straight one remains non-medullated. Similar cells with spiral fibres are met with in mammals.

§ 14. *Nerve-Fibres* are of various kinds, and often of complex composition.

(1) *Elementary Fibrils*.—The simplest fibres consist of very fine fibrils (*Fig. 1, 1*), which, in the fresh condition, as well as after staining with chloride of gold, present minute, more or less regular varicosities (Max. Schultze, Cohnheim). These fibrils may be observed in the terminal distribution of many nerves, as the stratum of the optic fibres in the retina, and the terminal distribution of the olfactorius, and in the ultimate distribution of the nerves over unstriped muscular fibres. They may also be observed in the brain, and in the grey substance

of the cord, as the finest subdivisions of the processes of the ganglion-cells.

(2) *Axis-Cylinder*.—The axis-cylinder is made up of a large number of elementary fibrils, arranged longitudinally (*Fig. 1, 2*). The longitudinal arrangement of the fibrils gives to the axis-cylinder a longitudinal striation; while minute granules are seen between these which appear to indicate that the fibrils are held together by a granular cement-substance. The unbranched process of the large multipolar ganglion-cells, and which from its continuity with the axis of the medullated nerve-fibres has been called the axis-cylinder process, forms the most exquisite example of a naked axis-cylinder (*Fig. 1, 12*).

(3) *Non-Medullated Nerve-Fibres*.—The fibres of Remak (*Fig. 1, 3*) consist of an axis-cylinder and a sheath, which, from its discoverer, has been called the sheath of Schwann. These fibres are also, from their colour, called grey fibres. The sheath of Schwann is a thin hyaline elastic membrane, and its internal surface is covered at regular intervals with oval nuclei. These fibres occur in the sympathetic system of nerves, and in the olfactory nerves; while the fibres of all nerves up to a certain stage of embryonic life and in most of the invertebrates are of this variety. The sheath of Schwann appears to be, like the sarcolemma of muscular fibre, hyaline and structureless; and, although nuclei are scattered on its internal surface, yet they do not seem to belong to this sheath, but to be independent structures, and the precursors of the next complication of the nerve-fibre.

(4) *Medullated Fibres*.—It may be supposed that the development of the non-medullated into the medullated fibres occurs somewhat in the following manner:—The nucleus on the interior of the sheath of Schwann is really not naked, but is surrounded by a layer of protoplasm, and may be said to represent a nucleated cell. Let us now attend to the changes which occur when a nucleated cell develops into a fat cell. The oily substance collects in the interior of the protoplasm, while the outer layer of the latter hardens into a distinct cell-membrane; and, as the oily contents accumulate, the nucleus is pushed towards the periphery, and the protoplasm is stretched so as to form a thin layer lining the interior of the membrane.



The layer of protoplasm becomes, indeed, so thin that its presence is apt to be overlooked. Instead of ordinary fat, we have only to suppose that a fatty material of very special character accumulates in the interior of the protoplasm, surrounding the nuclei interposed between the sheath of Schwann and the axis-cylinder, in order to account for the development of the medullated from the non-medullated fibres (Ranvier). As the protoplasm which surrounds the nucleus becomes distended with its oily contents, it insinuates itself between the sheath of Schwann and the axis-cylinder; and, after surrounding the latter, the free margins of the cell become fused. It is evident, therefore, the axis-cylinder will be surrounded, first, by a membrane and a thin layer of protoplasm, then by a more or less thick layer of oily material, and then by another thin layer of protoplasm and membrane, to the latter of which will be attached the nucleus, and surrounding the whole will be the structureless sheath of Schwann. This account of the development of the medullated from the non-medullated fibres has the merit of giving a pretty accurate account of the appearances presented by the former. Surrounding the axis-cylinder, which has been already described, is the medullary sheath, which appears as a thick, sharply outlined, doubly-contoured, fatty, semi-fluid substance (*Fig. 1, 6, b*). This substance coagulates very soon after death, and separates either spontaneously or under the action of various reagents into smaller or larger globular drop-like bodies (*Fig. 1, x*). When treated with perosmic acid, the medullary substance has been seen to consist of longer or shorter cylindrical sections, which are imbricated at their margins, although this arrangement is not observed in all fibres. Each of these sections contains a reticulum, in the meshes of which the fatty substance is embedded. When the reticulum is examined from the surface it gives a honeycomb appearance to the nerve; but when viewed in profile it appears composed of rod-like elements, which are the septa of the honeycomb seen sideways. A narrow space, which has been called the periaxial space, has been observed between the axis-cylinder and the medullary sheath. This space contains fluid albuminous cement substance, which coagulates under the action of hardening reagents, and then forms a thin granular membrane surrounding the axis-cylinder.

The nucleus surrounded by a smaller or larger film of protoplasm lies embedded on the outer surface of the medullary substance, and contains an intranuclear reticulum; while the surrounding protoplasm often contains pigment granules. The nucleus and surrounding protoplasm, along with the thin film which intervenes between the sheath of Schwann and the medullary substance, represent the outer wall of the cell from which the medullary sheath was developed.

(5) *Varicose Fibres*.—Some medullated nerve-fibres, especially in the central nervous organs and optic nerves, present more or less regular varicose thickenings (*Fig. 1, 4*). These varicosities are due to local accumulations of albuminous cement substance in the periaxial space, and not to a coagulation of the medullary sheath, as was once supposed.

(6) *Ranvier's Nodes*.—If, then, the medullary sheath is developed from the cells interposed between the sheath of Schwann and the axis-cylinder, it becomes interesting to enquire whether any trace can be discovered to indicate where two adjoining cells meet. The annular constrictions which appear at regular intervals in the course of the nerve, and which were first described by Ranvier, and consequently are called Ranvier's nodes (*Fig. 1, 8*), are generally accepted as proof of the cellular origin of the medullary sheath. Each node is due to an annular fold of the sheath of Schwann projecting towards the axis-cylinder; while there is an interruption of the medullary sheath corresponding to each constriction. The part of the sheath between each constriction is called an *interannular segment*, and it is important to notice that each of these segments possesses one, and only one, nucleus, which is situated near its middle (*Fig. 1, 6*). Ranvier has found a finely granular substance in the concavity of the constriction, of the nature of albuminous cement substance. This cement substance becomes deeply stained with nitrate of silver, which also penetrates more or less deeply into the interior of the fibre and stains the cement substance surrounding the axis-cylinder; so that, after staining with silver, the nerve-fibre presents a peculiar dark cross at the node (*Fig. 1, 8*), the longitudinal branch of the cross appearing longer or shorter, according as the staining has extended a greater or shorter distance on the surface of the axis-cylinder.



The nodes of Ranvier have probably an important function to perform with respect to the nutrition of the fibre, inasmuch as they permit the plasma to penetrate more freely to the axis-cylinder than could possibly take place if the medullary sheath were continuous.

Some medullated nerve-fibres, more especially efferent fibres, subdivide into two or more branches, the division taking place at a node of Ranvier.

(7) *Connective Tissue Cells*.—Elongated nuclei may be observed lying outside the sheath of Schwann (*Fig. 1, 6, d*). They are the nuclei of flattened connective tissue cells which are formed between the individual nerve-fibres, and constitute the cellular portion of the *endoneurium*. In isolated fibres these cells form a continuous endothelial membrane surrounding the fibres towards their terminal distribution, and they are closely connected with the lymph-spaces of the endoneurium, which will be subsequently described.

§ 15. *Functions of Ganglion-Cells*.—We may expect that the contrast exhibited in the structure of the different kinds of ganglion-cells is paralleled by a corresponding contrast in their functions. The first contrast which we notice is that of size. It is evident that a large size enables the cell to give rise to a powerful discharge of energy; and we may expect that the most powerful discharges will emanate from those cells which are in immediate relation with the outgoing currents, and that the smaller cells are in relation with the incoming currents, which require to be arranged and elaborated before being reflected outwards. This expectation is realised. The large cells are met with in the spinal cord in connexion with the efferent and the small cells in connexion with the afferent nerve-fibres.

The next contrast between the spherical and the caudate cells is, that the former do not possess a definite cell-wall, and have no definite connexions; while the latter not only possess a definite cell-wall, but have also, by means of their processes, extremely definite connexions with one another and with nerve-fibres. It is manifest, therefore, that the currents through the former must pass in a somewhat diffused manner, while in the

latter they will pass through very defined channels. The former, then, are adapted for the first elaboration of the incoming currents, and the latter for the final co-ordination of the outgoing currents. If the afferent currents are few in number, and if they are at the same time well organised in the race, they may pass more or less directly to the caudate, without the intervention of round cells. The actions to which the internal organs are subjected are uniform in kind, and relatively few in number. The stomach, for instance, passes through the same kind of actions day after day with great uniformity; its actions are also thoroughly organised in all animals; hence the afferent currents from it to the sympathetic ganglia pass directly into bipolar cells, and issue from these as efferent currents. The afferent currents ascending from the feet during locomotion are also few in number, simple in kind, and thoroughly organised in the race, and it is probable that these pass from the afferent fibres directly to the large motor cells, without previously passing through the small round cells of the posterior horns.

The next contrast I shall notice is one existing between the caudate cells themselves. Some are unipolar or bipolar, and others multipolar, with exceedingly ramified connexions. Some anatomists doubt the existence of unipolar cells; but we may expect to meet with the bipolar cells where the co-ordinations to be effected are few and simple, and the multipolar where the co-ordinations are numerous and complicated. We have already noticed that the movements of the internal organs are comparatively simple and uniform; and it is mainly in the ganglionic centres which preside over the functions of those organs that the bipolar cells are found; while the best examples of the multipolar cells are found in the anterior horns of the cord in direct relation with the nerves which convey efferent impulses to the organs of external relation, where numerous and complicated movements require to be effected.

The order of the development of the nervous system, structurally regarded, is from the round cell without membrane, to the caudate cell with membrane; and from the caudate cell with few and indeterminate connexions, to those with multiform



and defined connexions : just as the order, functionally regarded is from actions which are diffused and simple, to those which are less diffused and simple ; and from the latter, to those which are well defined, multiform, and complicated. This order is equally true, whether the progress of development is contemplated under the aspect of the transition from the lower to the higher animals, or from the initial to the adult stages of the higher animals ; or under the aspect of the process known as education in the higher animals.

Ganglion-cells contain a store of material possessing potential energy, which, on the application of a liberating force, becomes kinetic. The liberating force which renders the potential energy kinetic is termed a *stimulus*. The chemical process which underlies the transformation is probably of the nature of oxidation, since the blood, returning from the brain, for instance, is as venous as that returning from any other part of the body ; but we possess no direct evidence of the nature of this change. In reference to the liberating force, it may be noticed that the ganglion-cells do not appear to respond to the usual mechanical and chemical stimuli. By applying strong electrical stimuli to masses of ganglionic-cells, such as those of the cortical part of the brain, a response has been obtained from them in the form of muscular movements. These cells are, however, connected with each other by innumerable fibres, and there lie immediately beneath them large masses of nerve-fibres to which the currents, from the strength employed, must be conveyed ; hence it is extremely doubtful, to say the least, how far the muscular movements in such a case can be taken as evidence of the direct action of the current on the cells. When the ganglion-cell lies between two nerve-fibres, as those engaged in reflex action, the potential energy of the cell is set free by means of the already liberated energy of the stimulated fibre ; and the energy thus set free renders kinetic the potential energy of the second fibre. The cell largely increases the amount of energy rendered kinetic during the action, but the function of the cell in this case does not greatly differ, except in degree, from that of a nerve-fibre. But even in such a simple case the cell generally becomes the point of union of several fibres, and thus it helps to direct the disturbance it receives through one fibre into two

or more fibres, and becomes the active agent in giving a new direction to the current.

The manner in which the energy of the cells is liberated in automatic action is not easy to understand. It is probable that a great many of the actions regarded as automatic are of reflex origin. There is another way in which the energy of the automatic cell may be supposed to be liberated. The energy set free during one moment may perform the part of a liberating force the next moment on the store of potential energy, which is being constantly replenished from the blood, just as a fire, when once kindled, may be kept burning if supplied with combustible material. But the liberation of energy effected by this means would be continuous; while the liberation of energy in a stimulated nerve-fibre is interrupted or intermittent. It is quite possible, however, for a continuous liberation in a ganglion-cell to give rise to an interrupted or rhythmical stimulation of a nerve-fibre. Suppose that the energy liberated in the cell has to overcome a certain resistance before acting as a stimulus on a nerve-fibre, a certain tension must be reached prior to stimulation; and when the requisite tension is reached, a discharge takes place through the nerve. This discharge diminishes for a time the tension of the energy liberated in the cell; and, as it is probable that the molecules of the axis-cylinder have fallen during the discharge from an unstable to a stable equilibrium, the resistance to a second discharge through the fibre will be increased. The continuous liberation of energy within the cell soon raises the tension again; while by restorative processes in the axis-cylinder its molecules are once more replaced in their position of unstable equilibrium, and the conditions for a second discharge are quickly restored, to be again succeeded by the conditions of a second interval. When the resistance is great it will require a high tension to overcome it, and this implies that the liberation of energy must continue for a long time before the necessary degree of tension is reached, and that when the discharge takes place it will be a powerful one. Strength of discharge, then, involves length of interval between the discharges; or, in other words, the strength of every discharge of energy through a nerve is inversely proportional to its frequency.



§ 16. *Functions of Nerve-Fibres.*—The axis-cylinder, or more properly the elementary fibril, is the essential constituent of the nerve-fibre, and has been aptly compared to the *core* of copper wire strands in a submarine telegraph cable. The next most important constituent is the primitive sheath, which has been compared to the outer coating of rope of the cable. The last constituent, and therefore that which distinguishes the most highly differentiated fibre, is the medullary sheath, which has been compared to the layer of gutta-percha in a telegraph cable.

Glancing now at these three kinds of fibres, the most noticeable feature is, that all of them are adapted for conveying impulses in the direction of their length; but when several of the fibres of Remak are arranged side by side, free lateral diffusion of the currents will take place, while the primitive sheath of the non-medullated fibres will check this diffusion to a considerable extent, and it will be entirely prevented by the medullary sheath of the medullated fibres. We may expect, therefore, to find the first order of fibre when the function is of a very diffused character, the second when the function is less diffused, and the third when it is very defined, and when, consequently, any lateral diffusion, or irradiation, as it is called, would mar the effect. The fibres of Remak are mainly found in the intracranial portion of the olfactory nerve; and it is well known that, of all the special senses, the olfactory is the most diffused. The non-medullated fibres are met with in the sympathetic nerves, which preside over the actions of the organs of internal relation—actions which are much more diffused than those of the organs of external relation; while the medullated fibres alone are fitted to preside over the definite actions of the latter organs. Any lateral diffusion of the nerve-currents would entirely mar the definite and delicate movements of the hand required for writing; while some degree of this diffusion would appear to be necessary for that continuous and diffused contraction of the muscular coat of the bowels which causes peristalsis.

Nerve-fibres exist in the conditions of rest, activity, and death. In passing from one state to another their physical

properties undergo no perceptible change such as takes place in muscle; hence these different conditions cannot be distinguished from each other in nerve by simple inspection. A nerve is living if it possess *irritability*. The agents which evoke the activity of a nerve-fibre are, as in the case of the ganglion-cells, termed *stimulants*; while the property which nerve-fibres possess of transmitting the state of activity from one point to the next, is called their *conductivity*. When the irritability is not called into activity by any stimulus, the nerve is at rest. No doubt a certain amount of material exchange takes place in a nerve, as in other tissues, during a state of repose; but as nerves are almost destitute of blood-vessels, the material exchanges which take place in them must be slight. When the nerve-fibre gives evidence of evolving energy when acted on by a stimulus, it is in an active condition; and when it gives no response to the action of a stimulus, it is dead. Death of nerve-tissue is also recognised by the appearance of an acid reaction, and by certain electro-motor phenomena as well as by the loss of irritability.

(i.) *Irritability*.—The laws of irritability have already been considered in a general way; but we must now establish empirically the laws which influence the degree of nerve-irritability.

(1) Continued inaction of a nerve diminishes and finally destroys its irritability, and leads at last to fatty degeneration. It is very probable that the first effect of inaction is to increase the irritability.

(2) The irritability of a nerve, after it ceases to be connected with a living central organ, increases considerably at first, but afterwards diminishes, and finally disappears. When the separation is effected by transverse section, the process is accelerated, and it also takes place more quickly in the central than in the peripheral part of the nerve. In this case, some part of the effect is no doubt due to the mechanical stimulus of the section. If the cut nerve be allowed to remain in the body, it undergoes fatty degeneration. But if the cut ends be kept in apposition, they grow together after a time, and they have even been known to grow together when the cut ends were half an inch or more apart.



(3) Gentle stimulation of a nerve increases its irritability; over-stimulation diminishes it, and may destroy it at once (shock). Continued activity diminishes the irritability, and may gradually destroy it (exhaustion).

(4) Mechanical irritants, such as crushing or pinching, destroy the irritability. If the mechanical stimulus, however, is not too violent, the irritability is first increased, as occurs after section of a nerve with a sharp knife.

(5) A decided disturbance of chemical composition, such as is caused by desiccation and treatment with strong alkalis or acids, destroys the irritability. Certain agents absorbed into the blood, such as strychnia, first increase and then destroy the irritability of certain nerve-fibres, and probably also of some ganglion-cells. Curara lowers the irritability of the peripheral terminations of voluntary motor nerves; but Bernard found that a moderate dose first increases the irritability, which then becomes diminished and ultimately lost.

(6) The state of the nutrition of a nerve has a great effect on its irritability. If the nutrition is wholly arrested, the irritability disappears. But a nerve whose nutrition is merely defective discharges its energy more readily than one whose nutrition is perfect.

(7) Gradual withdrawal of heat diminishes, and gradual addition of heat (within certain limits) increases, the irritability. Too much heat lowers and may instantly annihilate it by inducing chemical change.

(8) If a constant galvanic current be passed through a portion of a nerve, it acts as a stimulus at the moment of making and breaking contact. When the current is passing through the nerve, the latter appears to be at rest, but its irritability is profoundly affected. This condition is called *electrotonus* or the *electrotonic condition*, but it would serve no useful purpose to enter on its consideration here.

(ii.) *Conduction*.—A particular nerve-fibre usually transmits its activity in one direction only—hence the distinction made between afferent and efferent fibres; but several circumstances might be mentioned which tend to prove that a nerve-fibre can conduct in both directions. It has been proved by experiment, that if a purely afferent nerve (gustatory) be

divided, and its central end be made to unite with the distal end of a divided motor nerve (hypoglossal), irritation of the former after the parts have been healed produces contraction in the muscles supplied by the latter.

Continuity of the nerve-fibre, especially of the axis-cylinder, is necessary for conduction. Lesions of the fibre from section or caustic, or even from a certain amount of pressure, interrupt conduction.

The transmission of the active condition from one end of a nerve-fibre to another occupies time. The average rate of conduction in human motor nerves is found by experiment to be 111 feet, or 33 metres per second; and in the sensory nerves to be 140 feet, or about 42 metres per second.

The velocity with which nerve-energy travels may be increased or diminished. The rule is that cold, the condition of anelectrotonus, and all conditions which lower the irritability, diminish the velocity; while heat, the condition of catelectrotonus, and all conditions which raise the irritability, increase the velocity of conduction.

Pflüger observed that the effect of stimulating a motor nerve is so much the greater the further removed the point of stimulation is from the muscle. He explained this effect by supposing that the active condition of a nerve accumulates strength in transmission, like the momentum of a falling mass, as an avalanche. It is now considered more probable that this phenomenon depends upon the increased irritability of the more distant parts of the nerve caused by section.

(iii.) *Stimuli*.—All mechanical impressions, as blows, pressure, section, etc., which cause alterations of the form of a portion of a nerve, act as stimuli while producing the change. Irritability and conductivity are destroyed if the nerve has been permanently injured. Agents which alter the chemical constitution of a nerve with a certain degree of rapidity, act as stimuli. Some substances, as ammonia and solutions of metallic salts, produce death so rapidly that the development of the stimulating effect is prevented. A sudden rise of temperature in a nerve also acts as a stimulus and causes the nerve to discharge its energy.

Variations in the intensity of an electric current stimulate



nerve-fibres, the stimulation being the more powerful the more suddenly variations occur. The variation generally employed is that produced by making or breaking a current through the nerve; but a sudden increase or diminution in the strength of a current also acts as a stimulus. The shocks of frictional electricity have a very powerful stimulating effect, since the currents it forms are extremely rapid in their appearance and disappearance.

§ 17. *General Theory of Nerve Functions.*—These, then, are some of the more important empirical laws of the functions of nerve-fibres, and of the mode of operation of those agents which evoke their activities. Let us now endeavour to reduce these laws to some degree of order. On comparing them with one another, the most obvious relation which exists between them is—that the mechanical, chemical, and thermal agents which act as stimuli when suddenly applied to a nerve, increase the irritability when gradually applied and in a moderate degree of intensity, and destroy it when suddenly applied beyond a certain degree of intensity; and it has just been seen that all conditions which increase the irritability of the nerve also increase the velocity of conduction. Such are the facts which require explanation.

All the functions of nerve-fibres are closely connected with the fundamental property of irritability. We have already seen that irritability depends upon the molecules of the protoplasm being in a condition of unstable equilibrium, and that the energy is rendered actual when the molecules fall to a relatively stable position. There can be little doubt that nerve-irritability depends upon the protoplasm of the axis-cylinder being composed of molecules in unstable positions, and that these are so connected that the movement or alteration of one leads to the movement or alteration of those in the immediate neighbourhood. The true nature of this movement is not known, but the theory most consistent with facts assumes that it is a chemical change, either of the nature called isomeric, or an oxidation, such as occurs when a train of gunpowder is ignited at one end. The slow rate at which the energy travels from one end of a nerve to another, in com-

parison with the speed of electric currents, shows that the two kinds of energy must differ essentially. The agents which act as stimuli set up this chemical change at one end of a nerve, and this is slowly propagated to the other end. When these agents are gradually applied, they act by placing the molecules in more unstable positions without causing them to move towards one another, so that a slighter degree of stimulus will subsequently induce the necessary chemical change; while the same agents, when suddenly applied in high intensity, will produce such a large amount of chemical change as to destroy the irritability. The following illustration may enable us to some extent to realise what occurs in a nerve-fibre during the transmission of its energy. We have seen that when matter possesses potential energy, a certain force called the liberating force is necessary in order to render the energy actual; or, in other words, matter under those conditions offers a certain amount of resistance to change, and the higher the resistance the stronger must be the liberating force. Suppose two books, each a foot in height, to be standing on end on a table, and that the one is half an inch and the other two inches in thickness; the slightest tap on the free end of the first will cause it to fall, while it will require a considerable blow to cause the second to fall. The resistance which the latter offers to change is greater than that of the former. And if we arrange two rows of these books placed on end at convenient distances, in such a way that when the first book of a row is made to fall it will strike the second and cause it to fall towards the third, and so on till the row has fallen with the books overlapping each other, it will at once be noticed that the row made up of the thin books falls much sooner than that made up of the thick books. Each book of the latter row offers a greater resistance to change, and not only is a greater force necessary to initiate the movement in the first book of the row, but the transmission of the movement from one book to the other is delayed. When the molecules of the axis-cylinders occupy relatively stable positions, the irritability of the nerve-fibres is depressed, a greater resistance is offered to change, and a stronger stimulus must be applied to them in order to awaken their activities. But not only must the initial liberating force be stronger, but it must be stronger



at each point of the conduction—hence the velocity of the conduction will be rendered slower; while the converse obtains when the irritability is increased.

§ 18. *Construction of the Nervous Tissues.*—It has been seen that on ultimate analysis the nervous tissues consist of cells and fibres, and we must now trace the relative positions occupied by these elements in the construction of the tissues. The general law of organisation is that unlike functions entail unlike structures; and inversely, that unlike parts assume activities of unlike kinds. On looking at a simple nervous system, the greatest contrast in structure is shown between certain knots termed ganglia and certain cords termed nerves, which connect one ganglion with another, or with different parts of the organism. Since these parts exhibit the widest structural contrast existing in the nervous system, they will also exhibit the widest functional contrast. The ganglia are composed of nerve-cells with thin connecting processes, held together by a fine connective tissue; whilst the nerves are composed of nerve-fibres arranged side by side in a bundle, also held together by a firm connective tissue, and the whole surrounded by a fibrous sheath. Functionally regarded, the ganglia are originators of motion, and to some extent conductors also, while the nerves are mainly conductors, although it is probable that they also are in some small degree originators of motion.

In the principal nervous centres of the higher animals, however, the ganglia, instead of forming knots, have come by approximation and fusion to form a continuous mass, which from its colour is called the *grey substance*; and the conducting fibres, instead of forming cords connecting two separate ganglia, also form a continuous mass, which from its colour is called the *white substance*. But, besides the central organs and the conducting apparatus, we must distinguish in a nervous system the peripheral terminations of the conducting apparatus—terminations which are specially adapted on the one hand for receiving impressions from environing agents and objects, and on the other for transmuting the molecular motions of the nervous system into the molecular motions of the work-organs. It is not intended to enter upon even the most general consideration of



the peripheral terminations of the conducting apparatus ; hence our subsequent remarks will be confined to the consideration of the conducting apparatus itself, and to the central end-organs of this apparatus.

The conducting apparatus, as already pointed out, consists of cords called nerves, and continuous masses called the white substance, both of these being composed of nerve-fibres, which usually remain unbranched in their whole course. Nerve-fibres are grouped into three classes with reference to the direction in which the energy passes through them—(I.) Afferent fibres, or those which convey impressions from the periphery to a nerve-centre ; (II.) Efferent, or those which convey impulses from a nerve-centre to a work-organ ; and (III.) Intercentral, or those which conduct between two nerve-centres.

(i.) *Afferent Fibres*.—I. The afferent fibres are further subdivided into—(1) those which minister to reflex action, that is, those in which the disturbances, conveyed by an afferent nerve to a centre, are immediately transferred to efferent nerves ; (2) sensory nerves, or those which convey impressions from the periphery destined to reach the highest nervous centre, which we shall subsequently see to be the organ of mind (they are called sensory, because any disturbance of them produces a sensation) ; and (3) inhibitory nerves, or those which restrain action. It is generally assumed that there are three distinct kinds of nerve-fibres, corresponding to these three functions ; but this is an assumption which is by no means proved as yet. The same system of afferent fibres may minister to the sensory and reflex functions, and it may depend upon various other circumstances, such as the relative resistance which the intercentral fibres concerned in the operation offer to the conduction of the impression, whether one or other action, or both of them, will ensue. The balance of evidence, however, appears to be in favour of the view that distinct fibres exist for these functions. But the existence of inhibitory nerves is very doubtful. No one doubts the existence of an inhibitory function ; the only question which arises is, whether the function is simple or compound. If the function is simple, there will be special fibres to minister to it ; but if it is compound, it will be a resultant of the action of nerve-fibres, or of nerve-fibres and cells engaged

in other operations. For instance, I throw the flexors of my leg and thigh into a state of rigid contraction, yet there is no movement of my leg, because I have at the same time thrown the extensors into contraction. The one action counteracts the other, and the leg is kept in a state of rest; but although the nerves which supply the extensors have been made to perform an inhibitory function, there are no special inhibitory nerve-fibres concerned in the action. But let us take an example from a more purely inhibitory function.

If the central end of the superior laryngeal nerve after section be stimulated by a gentle interrupted current, the respiratory rhythm is rendered slow, or stopped altogether in a state of expiration; while, if the central stump of one of the divided vagi be stimulated, the respiration, which from the division of the nerves had become slow, is quickened again. If the nerve has become exhausted by previous stimulation, further stimulation of the main trunk may cause the respiration to become slower, or even to stop. In accordance with these facts it is generally supposed that the superior laryngeal branch of the vagus contains inhibitory fibres, while the main trunk contains both accelerating and inhibitory fibres, the former of which largely predominate. It is very doubtful, however, whether there is any necessity to assume the existence of any fibres except the afferent fibres engaged in ordinary reflex action. The afferent impulses are first conveyed to an automatic respiratory centre in the medulla oblongata, a centre which possesses a rhythmic action whereby it emits complex co-ordinated impulses of inspiration and expiration. The afferent disturbances brought to this centre by the pneumogastrics modify its regulative impulses according to the requirements of the system. When, however, the afferent fibres are stimulated by a faradic current, the rhythm of the molecular disturbances conveyed by them may be so out of relation to the rhythm of the automatic centre, that fibres, which usually excite the centre, may now arrest its action. It is well known that two strings may be arranged in such a manner that each will produce a musical note when made to vibrate separately; that they may under one set of circumstances produce musical notes, which harmonise with one another, and under slightly different circumstances produce,



not musical notes, but a succession of beats with intervening periods of silence. It is quite possible, therefore, that the different effects obtained by stimulating the central ends of the divided vagi and their branches may arise from the altered rhythm of the afferent impulses acting upon an automatic centre already engaged in action.

But the most remarkable instance of inhibition is offered by the heart. It is found that if the peripheral portion of a divided pneumogastric nerve be stimulated for even a short time by a faradic current, the heart is immediately brought to a standstill, with its cavities flaccid and dilated. Here it is assumed that the pneumogastric contains cardio-inhibitory efferent fibres. There are good grounds for believing, however, that the fibres of the pneumogastrics which end in the heart terminate in local automatic ganglia; and therefore these fibres must be regarded as intercentral, and not as true efferent fibres. Hence the cardio-inhibitory action of the pneumogastric is susceptible of the same explanation which has been given of the respiratory inhibitory action—namely, that the action of the automatic ganglia is arrested by the loss of harmony between the rhythm of their action and the rhythm of the impulses conveyed to them by the artificially stimulated nerve. This explanation is much strengthened by the fact discovered by M. Onimus, that by duly regulating the rhythm of the galvanic shocks passed through the nerve in correspondence with the cardiac rhythm he could increase the number and power of the cardiac beats. The influence of the vaso-motor nerves in causing dilatation of the minute arteries is another striking instance of an inhibitory action. The general law, as discovered by Ludwig and Lovén, is that when an afferent nerve is stimulated there is reflected to the part, along its vaso-motor nerves, an influence by which its minute arteries are suddenly dilated; while an influence is sent to every other part of the body, which slightly contracts the arteries supplying them. There are good grounds for believing that arterial tone is maintained by local peripheral nervous mechanisms present in or near the small arteries, which are connected by intercentral fibres with the central mechanism in the cord. The function of these mechanisms, like the functions of the local automatic ganglia of the heart, may be



stimulated or arrested according as the rhythm of the impulses conveyed to them from the centres in the cord are in harmony or out of harmony with the rhythm of their own actions. Loss of arterial tone, therefore, does not require us to postulate the existence of special inhibitory fibres. Everyone is conscious of being able by an effort of the will to stop reflex movements; and when the brain of a frog is removed, reflex actions are developed to a much greater degree than in the perfect animal. If the optic lobes of a frog from which the cerebral hemispheres have been removed be stimulated by placing upon them a crystal of sodium chloride, it will be found that the generation of reflex impulses in the spinal cord is greatly interfered with—that is, the stimulation of the optic lobes has inhibited the reflex action of the cord. From these experiments it has been assumed that specific inhibitory centres exist in the optic lobes. But this assumption is by no means warranted by the facts. It is quite possible that waves of disturbance conveyed along afferent reflex nerves to certain motor cells, may be met by impulses conveyed down through the ordinary channels connecting these motor cells with the higher nerve-centres, in such a way that the one molecular disturbance will counteract the other. This is rendered more probable, since various other instances may be adduced to show that one nervous action interferes with the execution of another without anyone ever thinking of ascribing such interference to special inhibitory centres or fibres. If the toes of one leg of a brainless frog are dipped into dilute sulphuric acid at a time when the sciatic nerve of the other leg is being powerfully stimulated by a faradic current, it will be found that either the reflex withdrawal of the foot does not take place, or that the period of incubation is much prolonged. Goltz observed that, in the case of the dog, micturition, set up as a reflex act by sponging the anus, was at once stopped by sharply pinching the skin of the leg. From these, and many other similar examples which might be adduced, it is evident that two sensory impulses, arriving at the same centre by different paths, may interfere with each other in such a way that either the one counteracts the other, or the stronger current caused by the major disturbance drafts off that caused by the minor disturbance, so that

the specific effect of the latter does not take place. These considerations, therefore, tend to show that inhibition is not a simple but a derivative function; and that, although it may be correct to speak of an inhibitory action, this affords no warrant for assuming the existence of distinct inhibitory centres and fibres.

But if it is probable that this classification of afferent nerve-fibres errs on the side of redundancy, it is still more probable that it errs on the side of deficiency. The reflex afferent fibres lead to the peripheral ganglia and spinal cord, while the sensory conducting paths, whether consisting of continuous fibres or relays of fibres and cells, have their terminus in the cortex of the brain. But a large number of the afferent conducting paths terminate in the basal ganglia, and more especially the optic thalamus. The same set of fibres may, however, under one set of circumstances conduct impulses to the cord, and under another to the optic thalamus, so that there may be no necessity to assume the existence of distinct fibres for conduction to the latter ganglion. This remark will not, however, apply in the case of the cerebellum. Conducting paths must exist which convey impulses from the periphery to the cerebellum as well as to the cerebrum; hence we must assume the existence of cerebello-afferent as well as cerebro-afferent fibres, or rather conducting paths, inasmuch as we do not know whether these channels consist of continuous fibres or of relays of fibres interrupted by cells.

(ii.) *Efferent Fibres* are subdivided into—(1) Motor, (2) Secretory, and (3) Trophic Fibres.

(1) *Motor Fibres*.—Motor Fibres are of various kinds. Some of these fibres constitute the efferent branch of the reflex arc; others by their excitation restrain action, and hence are efferent inhibitory fibres. It is probable, however, that these fibres really belong to the intercentral variety, and are not true efferent fibres. Other fibres are connected with an automatic centre, from which they convey impulses to the periphery; hence they may be called automatic fibres. But some of the impulses which are conveyed by efferent fibres are derived from still higher centres, although it is doubtful how far we are justified



in assuming the existence of distinct efferent fibres for each of these separate functions. It is possible, for instance, that the efferent limb of a reflex arc may convey voluntary as well as reflex impulses. Leaving this question undecided, we may safely say that some efferent impulses issue from the basal ganglia, and the resulting action may then from its complexity be called compound reflex. Other actions are guided from the centre of the brain, and those alone have a right to be called conscious actions. These motor actions are of two kinds. One kind is in relation with the desires and appetites, and the other in relation with the will. What may be called the emotional actions originate in the centre and pass out through the basal ganglia and spinal cord; while the voluntary actions originate in circumscribed areas of the centre, called psychomotor centres, and pass each through fibres which connect the cortex with the spinal cord without being interrupted by the basal ganglia.

But this enumeration does not exhaust the different kinds of motor fibres, or rather the functions of those fibres. There can be no doubt that a large number of the movements of the body are guided through the cerebellum; hence it must be assumed that there are cerebello-efferent as well as cerebro-efferent fibres.

(2 and 3) The consideration of the secretory and trophic fibres may be deferred for the present.

(iii.) *The Intercentral Fibres* may be subdivided into (1) Commissural fibres, or those which unite ganglionic centres of the same order; (2) Centripetal fibres, or those which convey impulses from a lower to a higher centre; (3) Centrifugal fibres, or those which convey impulses from a higher to a lower centre. The words "centripetal" and "centrifugal" are generally used as synonymous with "afferent" and "efferent." Mr. Herbert Spencer was the first to use "centripetal" in the sense given to it here, and it will conduce to clearness if a corresponding meaning be given to "centrifugal." It must be remembered here that in practice it is not possible to draw any sharp line of distinction between the fibres which connect the periphery with the centres and those which connect the centres



with one another; hence it becomes impossible to apply the terms centripetal and centrifugal always in the way in which they are here defined.

§ 19. *Construction of a Nervous System.*—Let us now consider the relations which the nervous centres bear to one another, and to the organism at large; or, in other words, the manner in which the nervous tissues and mechanisms are put together to form a nervous system. We have already seen that unlike parts have unlike functions, and for every distinct part of the organism we may expect to find a distinct function, presided over by a distinct nerve-centre. On the other hand, when the parts have become structurally fused, the functions also have become fused, and we may expect to meet with a similar fusion of the nerve-centres. In the development of an organism the first structural contrast arises between the outer and inner surfaces, represented by the epiblast and hypoblast of the germ-vesicle. The most fundamental structural and functional differences will therefore be found to exist between the outer system of organs which react on environing agencies, and the inner system of organs which carry on sustentation; and we may expect to find the most fundamental structural and functional differences between the nerve-centres which preside over these two systems. The usual classification of the nervous system into cerebro-spinal and sympathetic acknowledges this distinction, since the first presides over the actions of the external, and the second over those of the internal organs.

But a third layer, called from its position the mesoblast, is formed between the epiblast and hypoblast, and this layer gives origin to all the parts of the body consisting of connective tissue, muscles, vessels, and nerves, as well as to the urinary and generative organs. The parts derived from this layer serve to connect the external and internal organs; and, in so far as the intermediate tissues subserve the functions of the external organs, their functions are regulated by the cerebro-spinal centres; and when they subserve the functions of the internal organs, their functions are regulated by the sympathetic: while, in so far as their function is intermediate between the external and internal organs, but partially independent of

them, they have an intermediate and partially independent nervous system, termed the vaso-motor.

The next structural peculiarity of the organism, which we shall notice, introduces us to likeness, instead of unlikeness, in the arrangement of parts, accompanied by a similarity in the distribution of the nervous centres. A plane, passing longitudinally through a man, and from front to back, would divide the body into two bilaterally symmetrical divisions. And what is true of the body, as a whole, is to the same extent true of the nervous system, since the same plane would also divide it into two bilaterally symmetrical parts. Now, it may be laid down as a general law, that when the actions of any part of an organism and its relations with other parts are few and uniform, there will be a corresponding simplicity and uniformity about its nervous connexions; and that, on the other hand, when the actions to be performed by a part and its relations with other organs are very numerous and complex, multiformity and complexity will characterise its nervous connexions. The bilaterally symmetrical viscera can act with a large amount of independence of one another. The kidneys, for instance, act simultaneously, because the blood conveys to them, at the same time, the agent which excites their functional activity, but the action of the one is in large measure independent of that of the other. It is different, however, with the external organs taken as a whole. The two sides of the body must move together, even when the incitement to action comes from one side only, and the actions of the limbs in performing a definite function, such as locomotion, must be duly co-ordinated. These differences of functional interdependence between the internal and external bilaterally symmetrical organs amongst themselves are represented by corresponding differences of structural connexions between the two sides of their respective nervous systems. The two gangliated cords situated on each side of the vertebral column, and which represent the sympathetic system, are connected transversely, only by a plexus of fibres and small ganglia; while the two sides of the cerebro-spinal system are fused practically into one bilobed ganglion. In the spinal cord the ganglionic substance of each lateral half is connected not merely by commissural fibres, but by a strand



of grey matter, which undoubtedly permits much more numerous and complex connexions to be formed between the two sides of the spinal cord than can take place between the symmetrically placed ganglia of the sympathetic.

The next structural peculiarity we notice is, that the body is made up of a number of segments placed end on end, and there is a corresponding distribution of the nervous centres. That this is the case with the greater part of the sympathetic is readily recognised. Each segment of the body is represented by a vertebra and its appendages; and each vertebra has a ganglion lying on each side, or two in front of its body, one for each lateral half. There are twenty-four true vertebræ, but there are not twenty-four pairs of sympathetic ganglia corresponding to these; because the three upper cervical on each side have become fused into one, while the two middle and the two lower cervical have respectively become fused into one. The sacrum consists of five vertebræ, which have become partially fused into one piece; and there are usually five pairs of ganglia corresponding to them, but their number is liable to variation; while the coccyx, although consisting at an early age of four pieces, is practically fused into one bone, and in front of it there is one, or at most two, ganglia. The cranial bones, according to some anatomists, represent three vertebræ, which have become variously modified and fused in the course of development; but the sympathetic ganglia corresponding to these cannot be separately traced. It is probable that they have become fused partly with the large upper cervical ganglion, and partly with the medulla, in order that the whole system may be brought into relation with the higher centres of the cerebro-spinal system.

But the internal organs in the different segments of the body are neither structurally nor functionally separate, and we may therefore expect that the ganglia in each will be connected with those of the segment above and below it; so that all of them will form a chain of ganglia, longitudinally as well as transversely connected.

But the functions of the internal organs are relatively simple. In the digestive organs, for instance, the same series of processes have to be gone through after every meal, varying only with the



quantity and quality of the food. And this simplicity of functional interdependence is represented by correspondingly simple interganglionic connexions. The main connexion between the ganglia is represented by a cord formed of a bundle of fibres passing down on each side of the vertebral column, and uniting the homologous ganglia with one another; and when the connexion requires to be closer, as when a large organ occupies several segments of the body, it is effected by the plexus already mentioned.

§ 20. *Fusion of Nerve-Centres.*—The relations between the different segments of the trunk and the different parts of the cerebro-spinal system are not easily made out in man. In the articulata, on the other hand, these relations are readily detected, since a bilobed nerve-centre is found in each segment of the body, forming a chain of ganglia connected longitudinally by a double cord. In these creatures, however, even the actions of the organs of external relation of each segment possess a considerable amount of independence of the actions of the external organs of other segments. In the higher animals the general actions of the external organs are closely dependent upon one another. The body must move as a whole; and, although the vertebral column maintains its segmented character, yet during locomotion it is kept rigid by muscles, especially in man, so as to be practically one piece. And corresponding to this fusion of the functions of the external organs there is a corresponding fusion of their nerve-centres. The nerve-centres of each segment are united with those above and below them, not simply by bundles of fibres, but by ganglionic substance. In the human cord, for instance, the grey matter of each lateral half is continuous from the lower end up, not merely to the medulla, but through the grey matter of the floor of the fourth ventricle, and that surrounding the aqueduct of Sylvius to the grey matter lining the third ventricle. By this means the ganglia of the segments have become so fused longitudinally and laterally, that the grey matter of the cord forms a continuous tube extending from the conus medullaris to the tuber cinereum. One consequence of this fusion of homologous ganglia is, that the parts of the cord which correspond to the different segments of

the body have undergone considerable displacement. The cord usually ends at the lower border of the body of the first lumbar vertebra, but the nerves which descend to pass out through the remaining lumbar intervertebral foramina, and through the anterior sacral and coccygeal foramina, show that the lower part of the cord presides over the functions of the lower segments of the body, although it has by the approximation and fusion of the homologous centres suffered considerable longitudinal displacement.

We have just seen that, when the actions of a part are numerous and complex, multiformity and complexity will characterise its nervous connexions; and we must now notice that along with multiformity and complexity of nervous connexions there must go increasing massiveness of nerve-centres. The large size of the cerebro-spinal nervous system which co-ordinates the numerous and complex actions of the organs of external relation, in comparison with the size of the sympathetic system which co-ordinates the simple and uniform actions of the organs of internal relation, may be mentioned as an illustration of this law.

Other examples of the law are met with on comparing different parts of these systems with one another; such, for instance, as the cervical and lumbar enlargements of the cord, where the complicated movements of the limbs are primarily co-ordinated, in comparison with the remaining portions of it, where the simpler actions of the muscles of the trunk are co-ordinated. But the most striking contrast in size exists between the cephalic and vertebral portions of the cerebro-spinal system—a contrast so remarkable that it deserves special examination.

One reason of the large size of the cephalic portion is that the impressions conveyed from the surface by the nerves of special sense are first co-ordinated by it; and since these impressions are much more numerous and complicated than those conducted by the cutaneous nerves, larger nerve-centres are required for their co-ordination.

As I sit in my study, I receive tactual impressions from the chair on which I sit, and from various other objects which surround me; but on looking out of my window the impressions received by my eyes are almost infinitely numerous and com-



plex. I see what I judge to be green fields, houses, horses, cattle, men, and women, and on looking up to the sky I am profoundly affected by an object which I know by indirect reasoning to be millions of miles from me. The centre, therefore, which co-ordinates the numerous and heterogeneous impressions conveyed by the optic nerves must be much larger than those which co-ordinate the comparatively few and uniform impressions conveyed by the cutaneous nerves. As a proof of this may be cited the fact that, in the lower animals, the first cephalic enlargement, termed the optic lobes, takes place in connexion with the central end of the optic nerves. The impressions conveyed by the other nerves of special sense are less numerous, and, with the exception of the auditory nerves, much less numerous than those conveyed by the optic nerves; but they are more numerous and complex than those conveyed by the cutaneous nerves, and, other things being equal, require larger centres for their co-ordination.

So far we have only spoken of single centres corresponding to the different segments of the body, and of the fusion of these centres into bilobed ganglia and continuous masses. But in the course of development superior centres arise, which co-ordinate and control the inferior centres; but before a superior can control inferior centres there must be a nervous connexion between them; hence the superior centre, which has to co-ordinate the actions of a large number of inferior centres, must be more massive than each of the latter. In the higher animals not only do we meet with compound co-ordinating centres, but we also meet with doubly-compound, and probably even trebly-compound co-ordinating centres, each of them increasing in massiveness according to its position in the ascending scale of complexity. Now, in the lower animals the cephalic extremity has to move foremost and to encounter dangers, and it therefore becomes the end to which the actions of the rest of the organism must be subordinated; hence the compound co-ordinating centres must necessarily be aggregated in this extremity. If, then, it is considered that not only the simple centres, which primarily co-ordinate the impressions conveyed by the nerves of special sense, but also the compound and doubly-compound centres, which co-ordinate all the impressions of external agencies on



the organism with one another, and determine the reactions of the organism as a whole to external agencies, are lodged in the cephalic extremity of animals, it will be at once apparent why the cephalic is so much more massive than the vertebral portion of the cerebro-spinal nervous system.

§ 21.—*Encephalo-Spinal System*.—These, then, are the main laws which regulate the construction of a nervous system, and before proceeding further it will be useful to obtain a general view of the leading features of the structure of the encephalo-spinal system. The ganglion-cells of this system are collected into five principal masses of grey matter:—(1) The cortex of the cerebral hemispheres; (2) the basal ganglia; (3) the cortex of the cerebellum; (4) the grey matter of the corpora dentata of the cerebellum, with which may be associated the olivary bodies of the medulla and the red nuclei of the tegmenta; and (5) the tubular mass of grey matter which extends from the tuber cinereum to the conus medullaris of the spinal cord. Seeing that the central grey tube of the spinal cord enters into connexion with the grey substance of both the cerebrum and cerebellum, the encephalo-spinal system may be subdivided into the cerebro-spinal and cerebello-spinal systems; and even if the latter of these two systems is to some extent subordinate to the former, it is largely independent of it, and consequently the two may be regarded as performing co-ordinate functions.

§ 22. *Cerebro-Spinal System*.—Let us return, in the first place, to the general structure of the cerebro-spinal system. The accompanying diagram from Landois' "Physiologie" is a schematic representation of the cerebro-spinal system. The cortex of the brain is represented by (C C); the basal ganglia are represented by (C s), the caudate nucleus of the corpus striatum; (N l) the lenticular nucleus; (T o), the optic thalamus; and V, the corpora quadrigemina. The tegmentum and crura are represented respectively by H and P. The crura are represented by P; while R represents a section of the spinal cord, and *h* W and *v* W the posterior and anterior roots of the peripheral nerves respectively. The central grey tube extends from P to R. The fibres (*c c*) and (*a a*) represent intercentral

FIG. 2.

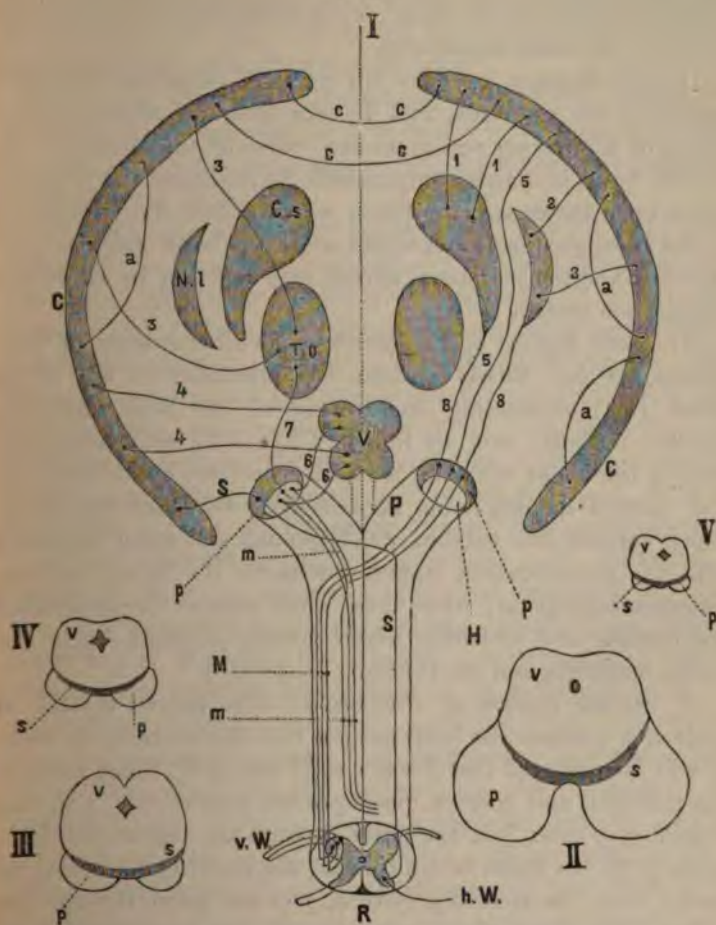


FIG. 2. *Schema of the Cerebro-Spinal System* (From Landois' "Physiologie").—  
 C, C, Cortex of the Brain; Cs, Corpus Striatum; Nl, Lenticular Nucleus;  
 To, Optic Thalamus; V, Corpora Quadrigemina; P, Crura Cerebri; H,  
 Tegmentum; p, Crusta; 11, Radiate Fibres of the Corpus Striatum; 22,  
 those of the Lenticular Nucleus; 33, those of the Optic Thalamus; 44,  
 those of the Corpora Quadrigemina. 55, the Pyramidal Tract. 66, Fibres  
 connecting the Corpora Quadrigemina and Tegmentum; m, their further course.  
 88, Fibres connecting the Corpus Striatum and Lenticular Nucleus with the  
 Crusta; M, their further course. S, S, course of the Sensory Fibres. R,  
 Transverse Section of the Spinal Cord; vW, Anterior, and hW, Posterior  
 Roots of the Nerves. a, a, associating Fibres; c, c, Commissural Fibres.  
 II, Transverse Section through the Crura Cerebri of Man on a level with the  
 posterior pair of the Corpora Quadrigemina (after Meynert); p, Crusta; S,  
 Locus Niger; v, the posterior pair of the Corpora Quadrigemina with the  
 Aqueduct of Sylvius. Similar sections from the Crura of—III, Dog; IV,  
 Monkey; V, Guinea Pig.



fibres, the former connecting points in one hemisphere with analogous points in the other; while the latter connect different points of the same hemisphere.

If with Meynert we take our point of departure from the cortex of the cerebrum, and if, like him, we regard as the object of all nervous action the projection of the image of the various forms of sensory impressions derived from the external world upon the cortex, the fibres which radiate from the latter to the basal ganglia, those which unite the basal ganglia with the cord, and the peripheral nerves, may together be called the "projection system."

(1) *Inner System of Projection.*—The inner system of projection, or the corona radiata, is represented in *Fig. 2* by fibres (1 1) connecting the cortex of the brain and the caudate nucleus; and by fibres (2 2), (3 3), and (4 4) connecting the cortex with the lenticular nucleus, optic thalamus, and corpora quadrigemina respectively. Of these the fibres which connect the corpora quadrigemina and optic thalamus with the cortex convey impulses towards the latter, and are therefore centripetal; while those which connect the cortex and the caudate and lenticular nuclei convey impulses from the cortex outwards, and are therefore centrifugal.

(2) *Middle System of Projection.*—The second system of projection connects the basal ganglia with the central grey tube. It will be observed that the fibres (7) and (6 6), which connect the thalamus and corpora quadrigemina respectively with the central grey tube, join the latter through the tegmentum (H); while (8 8), the fibres which connect the caudate and lenticular nuclei with the central grey tube, join the latter through the crusta. The fibres which pass through the tegmentum (6 6) and (7) are afferent, and are continued through the cord on the same side (*m*), but finally cross to the opposite side, near the level at which the peripheral nerve joins the central grey tube; while the fibres (8 8), which pass through the crusta, are efferent, and are represented in the figure as crossing over to the opposite side at the lower part of the medulla, and as being then continued onwards (M) in the opposite half of the cord. It is, however, very doubtful whether these fibres do cross in the lower part of the medulla. The fibres which



cross in the medulla appear all to belong to the system of fibres which connect the cortex of the brain directly with the central grey tube, and which will be immediately described under the name of the "pyramidal tract."

(3) *Outer System of Projection*.—The third system of projection is constituted by *h* W and *v* W, the posterior and anterior roots of the peripheral nerves, the former being afferent and the latter efferent.

(4) *Optic Radiations of Gratiolet*.—It will be seen, however, that the fibres which connect the cortex of the brain and the central grey tube are not yet exhausted. It will be observed that (S) and (5 5) represent fibres which connect these centres without being interrupted by the basal ganglia. The fibres represented by (S) issue from the convolutions of the occipital lobe, and converge towards the posterior portion of the internal capsule—the white substance which lies between the thalamus and caudate nucleus on the one hand, and the lenticular nucleus on the other—where they constitute one bundle of fibres which has been called the optic radiations of Gratiolet, after the anatomist who first described it. This bundle is frequently destroyed by disease of the internal capsule, when loss of feeling of the opposite side of the body results. These fibres ought to be represented as passing through the tegmentum, instead of the crusta, as in the figure; but whether they are continued onwards through the cord by fibres distinct from those which connect the cord and the thalamus is not known. There are no grounds for believing, however, that all these fibres cross in the medulla, as they are represented as doing in the figure. It is more probable that they cross to the opposite side, on a level with the point at which the peripheral nerve with which they are connected joins the cord.

(5) *Pyramidal Tract*.—The fibres represented by (5 5 M) issue from the convolutions of the middle lobe of the brain, and converge, on descending, until they form one bundle which occupies about the middle third of the internal capsule. These fibres proceed downwards without being interrupted by the basal ganglia, and occupy the middle third of the crusta (*Fig 5, p p'*), where they still form one bundle. In the pons these fibres are split up into several bundles by the transverse fibres of the middle

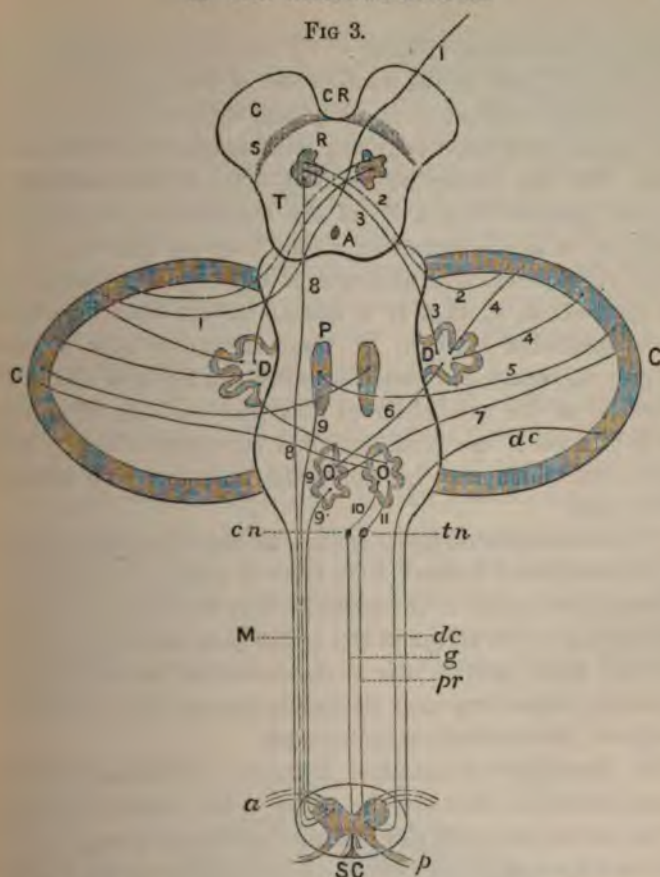
peduncles of the cerebellum; but they come together again to form one bundle in the medulla, where they constitute the anterior pyramid. At the lower part of the medulla the greater number of these fibres cross over to the opposite side, and are also directed backwards so as to form the posterior part of the lateral column of the cord (*Fig 4, p p'*), where some of these fibres are continued onwards to the lower end of the central grey tube. A small number of these fibres do not cross in the medulla, but are continued downwards in the cord in the anterior column close to the median fissure, constituting the column of Türck (*Fig 4, T*). Inasmuch as these fibres form the anterior pyramids of the medulla, they are called the *pyramidal tracts*. A transverse section of the crura cerebri of man is represented in *Fig. (2, II)*, on a level with the posterior pair of the corpora quadrigemina (*testes*), *p* represents the crusta, *s* the *substantia nigra*, *v* the corpora quadrigemina and the aqueduct of Sylvius, while the tegmentum lies between that and the *locus niger*. In *Fig. (2, III, IV, V)* similar sections are represented respectively of the crura of the dog, ape, and guinea pig. It has been observed by Meynert that the relative sizes of transverse sections of the crura in different animals bear a close relationship to the relative sizes of their brains. It would not be safe to draw any general conclusion from such a comparison as is here made with sections of the crura in different animals; but everyone must be struck with the enormous relative bulk of the crusta in the crura of man—a fact which is of very great significance when it is considered that the pyramidal tract passes through it, the fibres of this tract being almost certainly those which convey the voluntary impulses from the cortex to the central grey tube.

§ 23. *The Cerebello-Spinal System* is much less surely known than the cerebro-spinal, and the following schema must be taken to represent, along with a few certainties, a considerable number of conjectures.

The cerebellum consists of a body and three pairs of crura, by means of which it is connected with the rest of the encephalo-spinal axis. The grey substance of the cerebellum is



FIG 3.

FIG. 3. *Schema of the Cerebello-Spinal System.*—C, C, Cortex of the Cerebellum.

D, D, Corpora Dentata. O, O, Olivary Bodies. R, Red Nucleus of Tegmentum. P, Grey matter interposed between transverse Fibres of the Pons. CR, Crura Cerebri. c, Crusta. S, Substantia Nigra. T, Tegmentum. A, Aqueduct of Sylvius. 1, Fibres which connect the cortex of the Cerebrum and that of the Cerebellum on the opposite side. 2, 2, Fibres connecting the Cortex of Cerebellum and Red Nucleus of the opposite side. 3, 3, Fibres connecting the Corpus Dentatum of the Cerebellum with the Red Nucleus of the opposite side. 4, 4, Fibres connecting the Cortex of the Cerebellum with the Corpus Dentatum. 5, Fibres connecting the Cortex of the Cerebellum with Grey substance interposed between the transverse Fibres of the Pons on the opposite side. 6, Fibres connecting the Corpus Dentatum with the Olivary body of the opposite side. 7, Fibres connecting the Cortex of the Cerebellum with the Olivary body of the opposite side. 8, 8, Fibres connecting the Red Nucleus, 9, 9, those connecting the interposed Grey substance of the Pons, and 9', those connecting the Olivary body respectively with the anterior Grey Horn of the Spinal Cord. M, The Anterior Column of the cord through which the fibres pass. g, Column of Goll terminating in *cn* the Clavate Nucleus. 10, Arcuate Fibres connecting the Clavate Nucleus with the Olivary body of the same side. *pr*, The Posterior Root-Zone terminating in *tn*, the Triangular Nucleus. 11, Arcuate Fibres connecting Triangular Nucleus and Olivary body of same side. *dc*, *dc*, Direct Cerebellar Fibres ascending in the lateral column of the cord and connecting the vesicular column of Clarke with the Cortex of the Cerebellum.



found in the cortex (*Fig. 3, C C*) and in the dentate nuclei (*Fig. 3, D D*), and in the roof nuclei of Stilling, the latter of which we may neglect at present, both because they are small and because very little is known with respect to their connections. But the olivary bodies (*Fig. 3, O*) of the medulla and the red nucleus (*Fig. 3, R*) of the tegmentum are similar in structure to the dentate nuclei, and they are also closely connected respectively with the inferior and superior peduncles of the cerebellum, so that it is almost certain that they belong to the cerebellar system. To these structures may be added the grey substance interposed between the fibres of the middle peduncles of the cerebellum as they pass in front of and into the substance of the pons, which is represented by (*P*) in the figure, and which may be called the *anterior grey substance of the pons*.

(1) *Intermediate Ganglia of Cerebellum*.—The dentate nuclei (*D D*), the olivary bodies (*O O*), the red nuclei (*R*), and the anterior grey substance of the pons (*P*), bear the same intermediate relationship to the cortex of the cerebellum and the spinal cord that the basal ganglia bear to the cortex of the cerebrum and the cord; hence they may be briefly termed the intermediate ganglia of the cerebello-spinal system.

(2) *Cerebellar Projection System*.—Following then the nomenclature of Meynert, the fibres which connect the cortex of the cerebellum with the intermediate ganglia may be called the inner system of projection; those which connect the intermediate ganglia with the spinal cord, the middle system of projection, and the peripheral nerves, themselves form the outer system of projection for the cerebellum as well as for the cerebrum.

(3) *Cerebello-Spinal Conducting Paths*.—The inner system of projection is formed by the fibres which connect the cortex of the cerebellum with the dentate nucleus (4 4), those which connect the cortex with the red nucleus (2), the anterior grey substance of the pons (5), and the olivary body (7), all of the opposite side. To the same system of fibres belong those (3 3) which connect the dentate nucleus with the red nucleus, and (6) with the the olivary body of the opposite side. The middle system of projection is less known than the inner system. But

since the olivary body, the anterior grey substance of the pons, and the red nucleus of the tegmentum are closely connected with the anterior white columns of the cord (M) and their continuation through the medulla oblongata, pons, and crura, it may be inferred that the efferent impulses from the cerebellum are conveyed through these columns which are known to be motor in their functions; hence the efferent portion of the middle projection of the cerebello-spinal system is represented by 9 9' and 9'' which connect the red nucleus, the anterior grey substance of the pons, and the olivary body respectively with the anterior horns of the central grey tube.

(4) *Posterior Root Zones and Column of Goll.*—The afferent portion of the middle projection system still requires notice. Between the posterior roots (*Fig. 3, p*) of the spinal nerves and the posterior median fissure lies the posterior white column, which is divided into two portions; an inner wedge-shaped portion called the column of Goll (*Fig. 4, G*), the fibres (*Fig. 3, g*) of which ascend to terminate in a grey nucleus called the clavate nucleus (*Fig. 3, c n*); and an outer portion called the posterior root-zone (*Fig. 4, p r*), the fibres of which

FIG. 4.



FIG. 4. *Cord of Human Embryo at five months.*—*ah, ah'*, Anterior Horns of grey substance; *ph, ph'*, Posterior Horns of grey substance; *ar, ar'*, Anterior Root-Zones; *pr, pr'*, Posterior Root-Zones; *P, P'*, Pyramidal Fibres of lateral columns; *T*, Columns of Türek; *G*, Columns of Goll; *dc, dc'*, Direct cerebellar fibres; *c*, Anterior Commissure.



(*Fig. 3, p r*) also ascend (although probably in a succession of loops instead of continuously) to terminate at the lower end of the medulla in a grey nucleus termed the triangular nucleus (*Fig. 3, t n*). From the clavate and triangular nuclei fibres (*Fig. 3, 10, 11*) issue which end in the olivary body of the same side; so that through the medium of these nuclei and the olivary body, some at least of the fibres of the posterior column form a crossed connection with the cortex of the cerebellum; and these fibres very probably constitute the afferent portion of the middle system of projection of the cerebellum. The outer system of projection is formed by the nerves, and all that need be said respecting it is to remind the reader that the peripheral nerves must convey afferent and efferent impulses to and from the cerebellum, as well as to and from the cerebrum, and even if it be shown hereafter that the same fibres execute both functions, yet the existence of the former function should not be overlooked and merged in the more conspicuous phenomena which attend the latter function.

(5) *Fibres connecting the Cerebellum and Cerebrum.*—Two other conducting paths in connection with the cerebellum have still to be noticed. One of them springs from the cortex (*Fig. 3, 1, 1*), ascends in the superior peduncle, crosses over to the opposite side in the tegmentum, and ascends through the internal capsule to reach the cortex of the cerebrum, thus forming a straight connection between the two superior ganglionic centres. It is also probable that an interrupted connection exists between the two through the medium of the red nucleus and the optic thalamus. The decussation of the fibres of the superior peduncles of the cerebellum is represented in *Fig. 5, x*, which also shows the position of the red nuclei (*R R'*) of the tegmentum.

(6) *Direct Cerebellar Fibres.*—The second conducting path is one which issues from the central grey tube at the junction of the anterior and posterior grey horns, and the fibres (*Fig. 3, d c, d c*) of which, after passing outwards to reach the surface of the lateral column of the cord (*Fig. 4, d c*), ascend and pass through the restiform bodies to gain the cortex of the cerebellum. These fibres form a straight connection between the cortex of the



cerebellum and the central grey tube, without being interrupted by grey substance, and they are consequently called the direct cerebellar fibres. Their functions appear to be afferent.

§ 24. *Functions of Encephalo-spinal System.*—With respect to the functions of the encephalo-spinal system, all recent researches have, in my opinion, tended to confirm Mr. Herbert Spencer's hypothesis, "that the cerebellum is an organ of doubly-compound co-ordination in space, while the cerebrum is an organ of doubly-compound co-ordination in time." This hypothesis, originally stated in very general terms, has been adopted by Dr. Hughlings Jackson, and applied by him with wonderful fertility of resource to the explanation of the phenomena of disease.

FIG. 5.

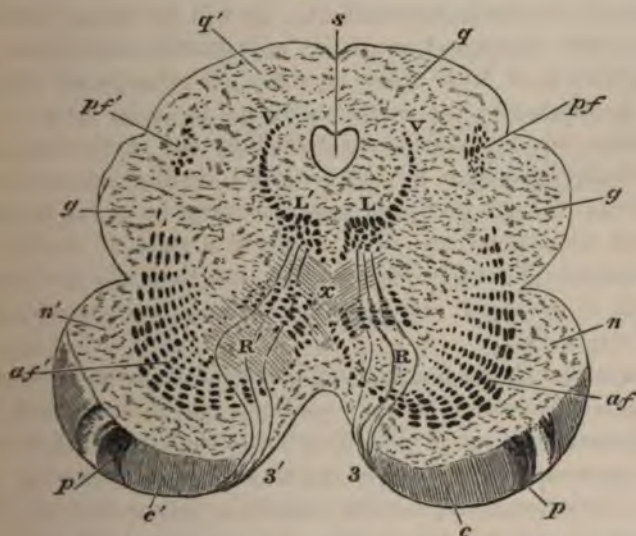


FIG. 5. *Crura Cerebri.*—Transverse Section of the Crura Cerebri on a level with the anterior pair of the Corpora Quadrigemina: from a nine-months human embryo. The dark portions represent Medullated Fibres. *s*, Aqueduct of Sylvius; *q, q'*, Anterior pair of Corpora Quadrigemina; *pf, pf'*, Fasciculi of Medullated Fibres proceeding to the anterior pair of Corpora Quadrigemina; *L, L'*, Posterior Longitudinal Fasciculi; *V, V'*, portions of these Fasciculi which join the posterior commissure of the third ventricle; *g, g'*, External Geniculate Bodies; *af, af'*, Anterior portion of Fillet; *n, n'*, Substantia Nigra; *R, R'*, Red Nuclei; *p, p'*, Pyramidal Tract; *c, c'*, Crustæ; *3, 3'*, Third pair of Nerves; *x*, Decussation in front of the Aqueduct of Sylvius, which is part of the interlacement of the Tegmentum.

According to this theory, the cerebellum regulates the muscular contractions necessary for the maintenance of all our attitudes in space, while the cerebrum regulates the contractions necessary to effect all the changes of attitude which are made in response to the successive impressions occurring in time. Now, so long as a particular attitude is maintained in opposition to gravity and other forces, the contractions of the various groups of muscles concerned must be *continuous* and in equilibrium with one another; while each change of attitude necessitates the overthrow of this equilibrium, involving the preponderance of the contractions of some groups of muscles over those of others, so that change of attitude involves *alternate* muscular contractions and relaxations. Speaking broadly, then, the cerebellum regulates *continuous* or *tonic* muscular contractions, while the cerebrum regulates *alternate* or *clonic* contractions. It will be seen, therefore, that every compound muscular adjustment necessitates the co-operation of both these organs. No change of attitude can be effected by the cerebrum except in so far as a certain attitude was previously maintained by the cerebellum, and no steady movement can be produced by the alternate contractions of some groups of muscles except in so far as other groups of muscles are maintained in a state of continuous contraction; hence it may be inferred that all the movements of the body are co-ordinated both in the cerebellum and cerebrum.

But although the functions of the cerebellum and cerebrum are to a considerable extent co-ordinate, yet it is manifest that the former must act in subordination to the latter. If animals only possessed the power of maintaining one unvarying attitude, they would not require capacities higher than those of inanimate objects; the degree of development to which an animal has attained is measured by its capacity of effecting multitudinous changes of attitude. Now, in effecting these changes, the alternate contractions under the guidance of the cerebrum must take the lead; and any change which is necessarily produced in the continuous contractions, although regulated through the cerebellum, must be in strict subordination to the action of the cerebrum.



§ 25. *Functions of Cerebro-spinal System.*—We have so far spoken of the functions of the cerebrum and cerebellum only; but it would be more correct to speak of the functions of the cerebro-spinal and cerebello-spinal systems, inasmuch as neither of the higher centres can act except through the medium of the spinal cord and peripheral nerves, which are common to both organs. But, however convenient it may be from a structural point of view to follow Meynert, and take our starting point from the higher centres, yet from a functional point of view it will be more convenient to start from the periphery. The order of the development of the nervous system is not from the cortices of the cerebrum and cerebellum to the central grey tube, but from the latter to the former; hence it is more philosophical to make the central grey tube rather than the cortices the starting point of our representation. According to this view the central grey tube, along with the peripheral nerves, constitutes a system of compound co-ordination in time (reflex action); the basal ganglia, when acting upon the central grey tube and peripheral nerves, form a system of compound co-ordination in time (instinctive action); and the cortex of the brain, when acting on the inferior centres, forms a system of doubly-compound co-ordination in time (conscious actions).

§ 26. *Functions of Cerebello-spinal System.*—The functions of the cerebello-spinal system may be similarly represented. The central grey tube, along with the peripheral nerves, forms a system of simple co-ordination in space as well as in time (*reflex tonus*); the intermediate ganglia of the cerebello-spinal system acting on the central grey tube and peripheral nerves form a system of compound co-ordination in space (the maintenance of unvarying attitudes); while the cortex of the cerebellum acting on the inferior centres forms a system of doubly-compound co-ordination in space (the adjustments of the tonic contractions of the muscles rendered necessary by changes of attitude).

§ 27. *Co-operation of Cerebro-spinal and Cerebello-spinal Systems.*—According to this theory, then, the multitudinous adjustments of the body, both in time and space, are regulated



by the combined action of the cerebrum and cerebellum acting through the spinal cord and peripheral nerves. The co-operation of these organs in the regulation of motor actions is, however, generally of an antagonistic kind. The cerebellum tends to maintain an unvarying attitude, while the cerebrum, in initiating a change of attitude, must act by overthrowing the balance of the muscular contractions which maintain this attitude. The overthrow of this equilibrium can be effected by the cerebrum in either of two ways, either *positively* by an increase of nervous impulses to certain groups of muscles, or *negatively* by arresting

FIG. 6.

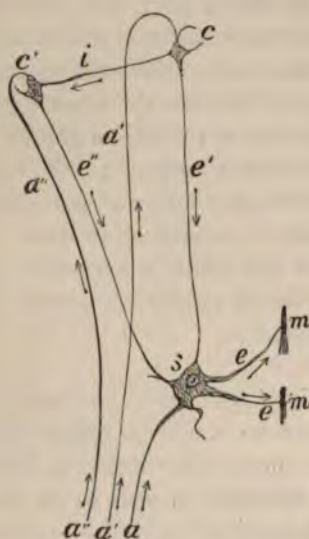


FIG. 6. Schema of Encephalo-Spinal Action. *s*, Motor ganglion cell of spinal cord; *c*, Ganglion cell of cortex of cerebrum, and *c'*, of cortex of cerebellum; *a*, *a'*, *a''*, Afferent fibres to the spinal cord, and to the cortices of the cerebrum and of the cerebellum respectively; *e*, *e'*, Efferent fibres from the spinal ganglion cell to *m*, *m'*, the muscles; *e'* and *e''*, Fibres from the cerebral and cerebellar cells respectively to the spinal ganglion cell. 2. Intercentral Fibre connecting the cerebral and cerebellar cells. The arrows indicate the direction of the conduction.

or inhibiting in the spinal centres the cerebellar influx to their antagonists. Now it is manifest that the latter method would be much more economical than the first, and consequently there is every reason to believe that the cerebrum does act largely by inhibiting the action of the cerebellum, although it is also certain that it must exercise a positive control over the various muscular contractions. The conjoint action of the central grey tube, the cerebrum and cerebellum, is represented in the accompanying diagram (Fig. 6) under the simplest conditions. A ganglion-cell of the spinal cord is represented by *s*, of the cerebrum by *c*, of the cerebellum by *c'*. The afferent conducting paths from the periphery to the spinal cord, cerebrum, and cerebellum are represented by *a*, *a'*, *a''* respectively. The efferent conducting path

between the cerebrum and spinal cord is represented by  $e'$ , between the cerebellum and cord by  $e''$ , and between the cord and muscles by  $e$ ; while  $m m$  represent the muscles themselves, and the arrows indicate the direction of the currents. Now, when an impression is made upon  $a$ , it is conveyed to  $s$ , and reflected through  $ee$  to  $mm$ , this constituting a simple reflex action. When an impression is made upon  $a''$ , the impulse is conveyed to  $c'$  and through  $e''$  to  $s$ , and through  $ee$  to  $mm$ , producing a continuous contraction of the muscles. But when an impression is made upon  $d$ , an impulse is conveyed to  $c$  and downwards, through  $e'$  to  $s$ . Now, the impulses conveyed through  $e'$  to  $s$  may produce, when of a certain degree of intensity, only an arrestive or inhibitory action on the impulses conveyed to  $s$  through  $a$  and  $e''$ ; while an additional degree of intensity enables it to pass through  $s$  and  $ee$  to  $mm$ , and to produce clonic muscular contractions. It is also probable that the cerebrum may exercise both an inhibitory and excitative action on the cerebellum through the intercentral fibres (*i*), which connect the centres directly with each other. This hypothesis was first stated by Dr. Hughlings Jackson, and he has since applied it, with his usual subtilty and generalising power, to the explanation of various pathological phenomena. As this is an exceedingly important theory, it will be as well to illustrate the actions of the cerebrum and cerebellum by reference to the muscular contractions necessary for the maintenance of the erect posture and for locomotion.

§ 28. *The Erect Posture.*—In the erect posture the weight of the body is borne by the plantar arches, and the body is maintained by a series of muscular contractions in such a position that the line of gravity falls within the area of the feet. In this position the line of gravity of the head falls in front of the occipital articulation; that of the combined head and trunk passes behind a line joining the two hip joints; that of the combined head, trunk, and thighs falls a little behind the knee joints; and the line of gravity of the whole body passes in front of the line drawn between the two ankle joints. This statement of the direction of the line of gravity shows that when the foot is made the surface of support, the body would



FIG. 7.

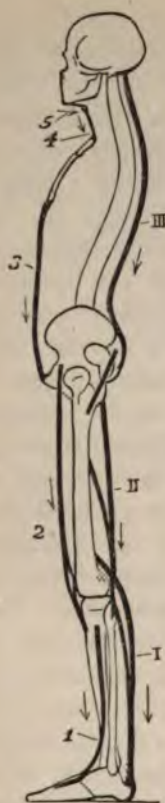


FIG. 7. (After Huxley.) Diagram illustrating the attachments of some of the most important muscles which keep the body in the erect posture. I, Muscles of the Calf. II, Those of the back of the Thigh. III, Those of the Spine, which tend to keep the body from falling forward. 1, the Muscles of the front of the Leg; 2, those of the front of the Thigh; 3, those of the front of the Abdomen; 4, 5, those of the front of the Neck, which tend to keep the body from falling backwards. The arrows indicate the direction of action of the muscles, the foot being fixed.

fall forwards unless prevented by contraction of the muscles of the calf (*Fig. 7, I*). "But this action," says Professor Huxley, "tends to bend the leg, and to neutralise this and keep the leg straight the great muscles in front of the thigh (*Fig. 7, 2*) must come into play. But these, by the same action, tend to bend the body forward on the legs; and if the body is to be kept straight they must be neutralised by the action of the muscles of the buttocks and of the back (*Fig. 7, III*)." It will be seen, however, that since the centre of gravity of the combined head and trunk falls a little behind the line joining the hips, the muscles of the buttocks, although strongly contracted in effecting the erect position, do not require to contract in order to maintain it.

The muscles of the calf, those of the front of the thigh, and the erector spinæ are therefore the most active muscles in maintaining the erect posture; and these are, according to the hypothesis, maintained in a state of tonic contraction, mainly by the cerebellum.

§ 29. *Walking*—At each step in walking there is a moment at which the body rests vertically on the foot of one leg (say the right), which is then called the "active leg." The other (left), which is now called the "*passive leg*," is at this time inclined obliquely, with the heel raised and the toe resting on the ground. The left leg, slightly flexed to avoid contact with the ground, is now swung forward like a pendulum, the length of the swing or step being determined by the length of the leg.



the left toe is brought to the ground, and the step is finished. The left leg, which was previously passive, now gradually becomes straight and rigid, and the body is moved forward on the left toe as a fulcrum; while the right leg, which was previously active, assumes an inclined position, with the heel raised and the toe resting on the ground, so that it is ready to swing forwards, and then once more to assume the rôle of activity, while its fellow becomes in its turn passive again. During the forward movement the centre of gravity of the body describes a curve, the convexity of which is upward; hence in successive steps the centre of gravity, and with it the top of the head, describes a series of curves, with their convexities upwards.

In standing on both feet the line of gravity falls between them, but in walking it must be alternately shifted from one foot to the other, in order to balance the body on the active leg. While the left leg, for instance, is passive and swinging, the line of gravity falls within the area of the right foot, and passes through the right lateral half of the pelvis, and as the left foot becomes active the centre of gravity is shifted to the opposite side, and the line of gravity passes through the left lateral half of the pelvis to the left foot. In walking, therefore, the centre of gravity describes not only a series of vertical but also a series of horizontal curves, so that the curve described by the head is composed of vertical and horizontal factors. In slow walking there is an appreciable time during which both feet are on the ground; the one being planted so as to become active before the other has ceased its activity. In fast walking this period is very short, the one leaving the ground the moment the other touches it; while in running there is an interval during which neither feet are on the ground.

Let us now attend to the muscles, the contraction of which effects the changes of attitude necessarily involved in walking. Suppose that we start with the right leg in the vertical position, with the line of gravity passing within the line of the right foot, and the left partially raised from the ground. The first indication of a forward movement must be effected by a contraction of the flexors of the foot on the leg, which, as the toe is fixed, bends the leg and with it the whole body forward. This contraction fixes the upper end of the tibia, the leg being bent

forward at an acute angle with the foot, and the femur is kept extended on the tibia by a rigid contraction of the muscles of the front of the thigh. The lower end of the femur and upper end of the tibia are now rendered fixed points, the line of gravity is rapidly passing forward from the middle of the foot to the toe, the weight is thus taken off the heel, and contraction of the muscles of the calf causes its elevation. But the line of gravity is now passing through the toe, in front of the knee, and in front of the centre of the hip joint, so that the muscles of the back of the thigh and those of the buttocks must contract strongly or the body would be flexed on the thighs, while the erectors of the spine must be sufficiently contracted to keep the different segments of the body in a rigid condition. It is manifest that, as soon as the line of gravity passes in front of the centre of the hip joint and through the toe, although muscular action may maintain the different segments of the body extended, no muscular action can prevent the body as a whole from falling forward on the toe as a pivot, and the body would fall unless the left foot were now in a position to be planted on the ground in front of the line of gravity, and ready to assume the rôle of the active leg. Before, however, the left leg can become active, the line of gravity must be transferred to the left foot, and before the right foot can be made to swing it must be shortened so as to avoid contact with the ground. These operations are so important as to require careful study.

§ 30. *The transference of the centre of gravity from the passive to the active leg* is largely effected by the contraction of the abductors of the thigh, and especially by the gluteus medius, contraction of which, the left thigh being fixed, causes the pelvis to rotate vertically on the hip joint; so that the centre of gravity, and with it the head, describes a curve to the left, with its convexity upwards, a movement which at the same time slightly elevates the pelvis and with it the hip joint of the opposite side. This slight elevation of the right hip joint not only transfers the centre of gravity to the left, but also increases the distance of the centre of movement (hip joint) of the passive leg (right) from the ground, and thus prepares for



the forward swinging of the right leg. The contraction of the abductors is accompanied by a contraction of their antagonists—the adductors, which not only gives steadiness to the pelvis but holds the latter in readiness to counteract at once any tendency to over-action on the part of the former, by which the line of gravity would be carried beyond the middle of the foot. The curve described by the head owing to contraction of these muscles would indeed be much greater than it is were it not compensated by contraction of other muscles. At the time that the abductors of the left leg contract, and thus rotate the pelvis, the centre of gravity, and head to the left, the erector spinæ of the right side enter into a somewhat additional contraction producing a compensating curve to the right, so that the head does not deviate to the left during the transference of the centre of gravity to the left foot to any thing like the extent that might be expected.

§ 31. *Swinging of the Passive Leg.*—It has just been said that when the left leg becomes active the pelvis rotates vertically on the left hip, so that the opposite hip joint is slightly elevated to an extent sufficient to take the weight of the body from the right toe; but inasmuch as the right foot is, at the time it is about to become passive, extended obliquely, with the toe depressed, while the left is placed nearly vertically, the former is much too long to swing past the other without touching the ground, and the slight vertical rotation of the pelvis just described does not give the requisite elevation for this purpose. In order to swing forwards, therefore, the right leg is still further shortened by flexion of its various segments on the body and on one another. The thigh is slightly flexed on the body, the leg on the thigh, and the foot on the leg. Of these movements the slight elevation of the toe caused by dorsal flexion of the foot is by far the most important and special; it is this movement which distinguishes the walk of the adult from that of the infant, the latter advancing the passive foot not by a pendulum motion, but by a voluntary effort in which the leg and foot are raised from the ground by flexion of the thigh on the body. It may also be mentioned that the adductors of the thigh manifest a very special action in assisting to cross



one leg over the other—an action which cannot be effected by the lower animals, or by the human infant, and hence these muscles must also be regarded as being in an especial manner under cerebral influence. The cerebro-spinal influence is therefore manifested in the active leg during locomotion by securing a strong contraction of the anterior flexors of the foot, and of the flexors of the leg on the thigh along with the abductors so as to fix and rotate the pelvis vertically; while it is manifested in the passive leg partly by contraction of the flexors of the thigh on the body, partly by contraction of the flexors of the leg on the thigh, and partly by flexion of the foot on the leg, the flexion in all these instances being probably due less to active contraction than to relaxation of the antagonist muscles. The transference of the line of gravity to the opposite leg also takes part in this action by removing the fixed point, from which the muscles act, from the foot to the pelvis.

§ 32. *The Act of Acquiring the Erect Posture.*—Now, if the changes of position which take place in walking are due to the predominance of cerebro-spinal over cerebello-spinal action, this is no less true with respect to the successive changes of posture requisite to raise the body from the recumbent to the erect posture. Suppose a man is lying in the prone position, and then gets up on his hands and knees. When the knees are raised by muscular action, so that the body is supported by the tips of the fingers and the toes, while the centre of gravity falls midway between the anterior and posterior extremities, this constitutes what I may call the *quadrupedal* position. In this position the toes constitute the fixed point for the posterior extremities, and the muscular strain rests upon the flexors of the phalanges and metatarsal bones, their contraction being necessary to maintain the rigidity of the plantar arch. The extensors of the foot on the leg must contract to prevent the tibia from being flexed on the foot, the extensors of the legs on the thigh must contract in order to prevent the thigh being flexed on the leg, and the extensors of the body on the thigh must also contract in order to prevent the former being flexed on the latter. A further contraction of these same muscles drags the centre of gravity of the body upwards and

backwards, the weight is taken off the anterior extremities, and the body assumes the semi-bipedal posture in which the line of gravity passes between the feet in the line which joins the toes, considerably in front of the line which joins the ankles, behind that which joins the knees, and in front of that joining the hip joints. It is manifest that the contractions of the muscles of the sole, those of the calf, front of the thigh, those of the gluteal region and the erectors of the spine, must largely predominate over their antagonists in order to maintain this position, and that this predominance must be maintained until such time as the heel touches the ground, when the line of gravity passes from the toes to the centre of the plantar arch, and behind the line joining the centre of the two hip joints. The vertical position is then maintained mainly by means of the bones and ligaments, aided only by a slight degree of muscular contraction. Now, the bipedal erect posture has only been attained from the recumbent position by passing through an infinity of intermediate postures; and according to the hypothesis the cerebello-spinal system has had to maintain each posture attained by striking a balance between the tensions of the extensors and flexors of the body, the latter being aided by gravity; while the cerebro-spinal system continually changes each attained posture by overthrowing this balance in favour of the extensors. In passing from the bipedal erect posture through the semi-bipedal and quadrupedal to the recumbent posture a reverse process takes place, the cerebro-spinal system, at each new position acquired, inhibits the action of the cerebello-spinal system on the extensors, so that contraction of the flexors assisted by gravity is allowed gradually to predominate.

But if this hypothesis of the joint, although opposite, action of the cerebrum and cerebellum acting through the spinal cord be true at all, it must be accepted in its fullest extent. If, for instance, it be true that the passage from what I have called the *quadrupedal* to the *bipedal* erect posture in the human subject is due in the individual to the predominance of cerebral influx to the extensors over their antagonists this is no less true with respect to the race. In the gradual development of man from the lower animals the same forces have been at work. From the semi-bipedal position assumed by a dog attempting



to stand on its hind legs, and the imperfect bipedal attitude of the monkey to the perfect bipedal erect posture of man, the transition must have been effected by the gradual predominance of the extensors over their antagonists through cerebro-spinal influence. In all these processes it will be seen that the flexors of the body are aided in their action by the force of gravitation, while the extensors have to overcome this force by their action; hence the latter must be capable of much more powerful contraction than the former, and are consequently more liable to have more powerful discharges sent to them both from the cerebro-spinal and cerebello-spinal systems.

When, therefore, both the extensors, and flexors of the head, trunk, and lower extremities are contracted to their utmost capacity, the action of the former must predominate over that of the latter; so that the segments of the lower extremities will be extended upon one another, and the body will be arched with the concavity directed backwards, as occurs during the paroxysms of tetanus. But the hand being mainly an organ of prehension, the principal function of the anterior limbs of man is to pull objects towards the trunk, the latter being the fixed point during their activity; hence the flexors of the upper extremities must be more strongly developed than the extensors. And when the hands become fixed, as in climbing, the most powerful contractions are obtained when they are in a position to drag the body towards the fixed position, and not, as in the case of the lower extremities, when the body is thrust upwards and away from it; hence, when the muscles of the upper extremities are contracted to their utmost capacity, flexion will predominate over extension.

§ 33. *Fundamental and Accessory portions of the Nervous System.*—Before leaving this portion of our subject I should like to establish one more distinction. Structure being the correlation of function, the multiplicity and complexity of the movements which distinguish man from the lower animals must be accompanied by a corresponding degree in the intricacy and variety of the structural arrangements of his nervous system. The main movements which distinguish man from the lower animals are those concerned in attaining and maintaining the



erect posture, the varied movements of the hands as organs of prehension, the movements of voice and articulation concerned in speech, and those which are active in the production of facial expression. All these movements must, therefore, be represented in the human nervous system by structural arrangements superadded to those which man possesses in common with the highest of the lower animals. Indeed all the complex movements first mentioned are acquired considerably after the birth of the human infant, and we may consequently expect that the structural arrangements corresponding to them either do not exist at birth or exist only in an embryonic condition.

The portions of the nervous system which man possesses in common with the lower animals, and which are well developed in the human embryo at nine months, I shall call the *fundamental* part; and the portions which have been superadded in the course of evolution, which differentiate the nervous system of man from that of the highest of the lower animals, and which are either absent in the human embryo, or exist only in an embryonic condition, I shall call the *accessory* part of the nervous system.

The fundamental portion of the human nervous system co-ordinates the fundamental functions which man possesses in common with the lower animals; but the accessory portions can only be said to regulate the accessory functions in a peculiar sense. The accessory structure constitutes indeed a new complexity of mechanism superadded to that already existing, a complexity rendered necessary for the regulation of the intricate and multiform actions which distinguish man from the lower animals. In the development of the accessory system, the small round cell and the non-medullated fibre appear at a comparatively late period in the development of the embryo, and the presence of these simple elements is regarded as the structural counterpart of a new modification or specialisation of function. Specialisation of function has hitherto been connected with the gradual development of medullated from non-medullated fibres, and of large caudate from small round cells; but now it appears that specialisation of function is to be connected with the development of embryonic cells and fibres. There is, however,

no contradiction between the two statements. The embryonic cells and fibres of the accessory system do not of themselves indicate any specialisation of function. These cells and fibres are, indeed, mere complications of an already existing mechanism, and this alone entitles them to be regarded as true indicators of a newly-acquired specialisation of function; they are, in short, mere modifications of an already existing structure corresponding to newly-acquired modifications of previously existing muscular adjustments. There can be no doubt that the fundamental and accessory portions of the nervous system will be so mingled together that it will be almost impossible to separate the two; but whether they can be distinguished from one another morphologically or not, the mental distinction is a valuable one, and it is important to remember that in man both the cephalic ganglia, the central grey tube, the conducting paths which connect them with one another, and even the peripheral nerves themselves, *must* contain fundamental and accessory cells and fibres.

§ 34. *The Law of Evolution.*—We have now passed very rapidly and very imperfectly under review the fundamental laws of nervous structure and function. We have seen that the nervous system consists essentially of cells and fibres, that the cells are first small, round, and uniform, that they gradually become large and assume numerous processes, that the fibres are at first small fibrils which together form larger fibres, that these become complicated by assuming an elastic sheath, and still further complicated by assuming a second sheath of a very special character. We have seen how the fibres come to be packed together to form white cords and the cells to form small masses of grey substance termed ganglia; how the cords integrate to form thick masses of white substance and the small ganglia to form masses of ganglionic grey substance, and how this continuous process assumed a still more complicated form when some of the ganglia became subordinate, while others exercised superordinate functions. The whole of the intricate processes here described illustrate the one great law of evolution. That law may be described as a progressive integration both of structure and function, during which there is a passage



from the uniform to the multiform, from the simple to the complex, and from the general to the special. During the evolution of the nervous system of man the fundamental portion is first developed. The nervous system of man is at first similar to that possessed by all animals which possess a nervous system, or at any rate all those which are sufficiently elevated to possess a spinal cord; but as development proceeds the nervous system of man becomes gradually differentiated from that of an ever-increasing number of the lower animals, while still maintaining a general likeness to the nervous system of the higher animals up to the time of birth. This, then, constitutes the *fundamental* portion of the nervous system of man; but after birth the *accessory* portion, which up till this time only appears in a rudimentary condition, now undergoes progressive development, and the nervous system of man becomes gradually differentiated from that of all other animals. It will thus be seen that the fundamental portion is first developed, and that the superaddition of the accessory portion greatly increases the multiformity, the complexity, and the speciality of the human nervous system, and it is consequently the latest product of its evolution.

§ 35. *Law of Dissolution.*—We must now proceed to regard the phenomena of the structure and function of the nervous system from a new and opposite standpoint. We must watch the cells lose their processes, and from the multiformity of the caudate cells with numerous processes pass to the uniformity of the round cells destitute of processes: we must observe the fibres losing their medullary sheath, then their elastic sheath, and finally the axis-cylinder itself becoming disorganised, so that the nervous tissue gradually gives place to a simple and uniform connective tissue: we must observe accompanying this process a corresponding loss of function, in which the complex movements that characterise health become difficult or impossible: in one word, we must trace the records of a process in which the progressive integration of evolution gives place to a progressive disintegration, during which the phenomena of structure and function, instead of passing from the uniform to the multiform, from the simple to the complex, and from the



general to the special, manifest a reverse tendency, of passing from the multiform to the uniform, from the complex to the simple, and from the special to the general. The law which governs this process is the law of dissolution, and it is the great law which regulates the phenomena of disease of the nervous system, just as evolution is the great law which regulates its growth and development.

It is scarcely necessary to add that the phenomena of evolution manifested in the growth and development of the organism are exceedingly gradual and continuous, and consequently the operation of the law of evolution can very readily be traced. Disease being, on the other hand, often sudden and violent in its onset, striking at times at the fundamental, at other times at the accessory portions of the nervous system, now producing its baneful influence at one stroke, again acting fitfully, and, only on rare occasions, in a gradual and progressive manner; it may be inferred that the operation of the law of dissolution can never be so clearly traced amongst morbid phenomena as that of evolution in the development of the organism. Nevertheless, there are some diseases of the nervous system which are gradual in their invasion and progressive in their course, and in them the operation of this law is clearly visible, and it is astonishing how glimpses of the law may be obtained even when the disease is sudden in its onset and rapid in its progress. One important corollary may be drawn from what has been said: that as the *accessory* portion of the nervous system is the last to be developed, it is the portion which is most liable to become diseased. Several reasons might be given why this should be the case, but these will appear in the subsequent pages. It will suffice at present to say that the accessory portion, from the late period of its development, is less stable than the fundamental portion, and that its necessarily frail structure will render it more liable to suffer both from accident and the inroads of disease.

## CHAPTER II.

## GENERAL ETIOLOGY.

THE causes of disease of the nervous system are the same as those of disease of any other part of the body; hence they do not require to be discussed at any great length at present. Whatever injures the protoplasm of any of the other cells of the body will injure the protoplasm of the cells of the nervous system; and whatever crushes or ruptures the cell wall, the intercellular substance, and fibres of other cells and tissues will have a similar effect upon the cell walls, the processes, and fibres of the nervous system. But a tissue which is so highly specialised as the nervous tissue will be specially acted upon by common causes; hence it is necessary to allude briefly to a few of the more ordinary causes of nervous disease. Various classifications of causes might be adopted, but the most convenient one is that which divides them into (1) Intrinsic and (2) Extrinsic: the former depending upon the individual in whom the conditions of disease are inherited or acquired, and the latter embracing the external incident forces which induce disease.

## § 36. INTRINSIC CAUSES.

(1) *Hereditary Predisposition*.—Some individuals inherit a predisposition to certain diseases of the nervous system. The predisposition to a particular disease may be *special and direct* or *general and indirect*. There suffers, for instance, from neuralgia, at a particular time of life, a lady whose mother had similarly suffered at a corresponding age. In this case the transmission of the affection is direct from mother to daughter, and



the transmitted disease is limited to a particular affection. But the inheritance is not only direct and special, it is also *immediate*; inasmuch as the disease is supposed to be transmitted from parent to child. In a large number of diseases, however, the inheritance is *remote*, the transmitted affection being derived not immediately from the parent, but from a grandparent, or a still more remote ancestor. The phenomena of *atavism* or *reversion*, as the remote inheritance is called, are, indeed, very conspicuous in the transmission of a large number of hereditary nervous diseases.

In other cases, however, the disease is not directly inherited, but an unstable nervous system is transmitted in which either neuralgia or some other disease of the nervous system is readily induced by slight external causes. In such cases a *neuropathic* tendency or a *neurotic disposition* is transmitted, which renders the nervous system exceedingly vulnerable. In such cases the tendency to any particular disease is *indirect*, and it is also *general*, inasmuch as one member of the family may suffer from neuralgia, another from chorea, paralysis, hysteria, epilepsy, or insanity, while others manifest a tendency to uncontrollable alcoholic excesses. The predisposition of nervous disease is still more *indirect* at other times. One man dies of disease of the brain, as his father died before him, at a particular age, but it is because both have inherited gout which has induced early arterial degeneration, which in its turn has ended in rupture of a vessel in the brain, resulting in sudden death. In another family several children in succession die in the midst of convulsions followed by coma, but it is because they have inherited a strong tendency to tubercular disease, and not from inherent weakness of the nervous system.

(2) *Age*.—The age of the patient exercises a powerful effect upon the predisposition to certain diseases. Some diseases of the nervous system are peculiarly liable to occur in childhood. So much is this the case in a certain form of spinal paralysis that it has been called infantile spinal paralysis, even although essentially the same disease occurs in adults; and it is notorious to everyone how very liable children are to be attacked with convulsions in comparison with adults. Other diseases, like hysteria and probably also neuralgia, very gene-



rally manifest themselves for the first time during the period of sexual development, while others are apt to come on during the period of sexual decline.

(3) *Sex*.—Females are more liable to certain affections than males, while the converse of this rule also holds good. Hysteria, for instance, although by no means limited to the female sex, occurs with such relative frequency in women that it was at one time supposed to be always due to some uterine derangement. Trigeminal neuralgia is much more frequently met with in females than males; while sciatica, on the other hand, occurs more frequently in males, probably owing to their being more exposed to its exciting causes.

(4) *Race*.—The influence which race exerts in the production of nervous diseases is not well known, but there can be no doubt that the civilised races of mankind are more liable to nervous affections than the less civilised races. And this is only what might be expected. The complex conditions of civilisation require the organisation of a more and more complex nervous system in the individual living amongst these conditions; and during the development of this complexity the mechanism is apt to break down at some part.

(5) *General Nutritive Disorders* produce a strong predisposition to nervous disease. All diseases attended with anæmia and cachexia depress the nutrition of the nervous system; hence the deleterious effect of loss of blood, chronic disturbances of digestion, and severe and protracted acute disease.

(6) *Sexual Excesses and Irregularities* exercise a very depressing effect upon the nutrition of the nervous system, and are consequently powerful predisposing causes of various nervous affections, such as neuralgia, various organic diseases of the spinal cord, hysteria, hypochondriasis, and even the graver psychoses. The effects of *onanism* are generally supposed to be more injurious than those which follow excessive natural indulgence, although this has not yet been proved beyond doubt. There can be no doubt, however, that frequently repeated onanism, practised for a succession of years, and associated, as such practice generally is, with habitual pollutions, must strongly predispose to grave nervous diseases.

## § 37. EXTRINSIC CAUSES.

(1) *Traumatic Influences* are the simplest and most direct cause of nervous disease. Wounds, contusions, lacerations of every variety, may injure the peripheral nerves, the spinal cord, or the brain, and give rise to the most complicated sensory motor, vaso-motor, secretory, and nutritive affections, according to the extent and locality of the lesion.

(2) *Slow Compression* of nervous tissues by pathological growths may also gradually injure any part of the nervous system, and thus give rise to various combinations of symptoms.

(3) *Direct propagation of neighbouring morbid processes* also frequently gives rise to severe affections. The transverse neuritis, which is set up as a sequel of disease of the vertebrae, may be mentioned as an example.

(4) *Exposure to Cold* is a frequent cause of disease of the nervous system; and the injurious effect of cold is greatly increased when it is combined, as is frequently the case, with excessive fatigue, want of nourishment, or other depressing circumstances. Severe disease of the nervous system, like tetanus, for instance, is very apt to arise in military campaigns during winter, when soldiers are often exposed to extreme cold after the heat, excitement, and fatigue of a great battle. Exposure to cold and damp, with insufficient food—conditions which are only too frequently combined in the cellar livings and back-slums of our large towns—is a prolific source of disease of the nervous system.

(5) *Disturbances of Circulation*, caused by the suppression of the menses or of hæmorrhidal discharge, arterial fluxion, and venous congestion, vaso-motor disturbances, embolism, thrombosis, atheroma of the arteries, and various other conditions, may be reckoned amongst the exciting causes of nervous disease.

(6) *Excessive exertion*, followed as it is by exhaustion, is a powerful exciting cause of disease of the nervous system, and becomes all the more powerful when it is combined with deficiency of food and exposure to cold and damp.

(7) *The local development of various infective diseases*, such as tuberculosis and syphilis, is a frequent cause of severe



disease of the nervous system. It is probable that in both these diseases the vessels and their adventitiæ, along with the connective tissue or neuroglia, are the primary seats of disease, and that the affection of the nervous elements is purely secondary; but the symptoms do not differ from those which would be produced by a primary affection of the nervous elements.

Grave diseases of the nervous system often arise in connection with the exanthemata, typhoid, and other continued fevers, pneumonia, and other acute inflammations, or as the result of malaria.

(8) *Chemical Poisons* of various kinds cause disease of the nervous system, many acting upon nervous tissue more or less directly, while others appear to influence it indirectly. Among the poisons which produce the most deleterious effects upon the nervous system are strychnine, arsenic, phosphorus, mercury, and lead. The human organism has become, through successive generations, so adapted to the use of ethyl alcohol as an article of diet, and as a means of increasing social enjoyment, that it probably now exercises the least deleterious influence on the system of any chemical poison of the same class. But notwithstanding this, the excessive consumption of alcoholic drinks, which is so common amongst all classes of society, renders ethyl alcohol—the active ingredient of these beverages—if not the most potent, at least one of the most important and widespread of the causes of disease of the nervous system.

Chemical poisons are being constantly generated in the system, as the result of normal disintegrative processes; but under healthy conditions these are eliminated so rapidly by the excretory organs that they do not accumulate in sufficient quantity in the blood to injure the nervous tissues. Where, however, the normal excretory processes are diminished or arrested by disease, or when an undue quantity of those poisons is generated, serious nervous disorder results. It is not, therefore, surprising to find that the diseases of the lungs, kidneys, and of the other excretory organs are frequently accompanied by grave nervous symptoms. The local application of acids and other powerful escharotics injures the nervous tissues, as well as the other tissues of the body; and these agents are not unfrequent causes of disease of the nerve trunks, which, from



their position, are more exposed to such injuries than the nerve centres.

(9) *Local Irritation and Disease of the Viscera*, such as chronic affections of the kidneys and bladder, chronic dysentery and other intestinal disease, diseases of the uterus and its appendages, peripheral lesions of nerves, and chronic joint affections, are often attended with spinal paralysis, which was at one time thought to be always of reflex origin, but in many cases this spinal affection is caused by an ascending neuritis having its starting point in the affected organ.

(10) *Psychical Disturbances* are a prolific source of disease of the nervous system, and it is probable that as civilisation advances these causes will exercise a more and more predominant influence in the production of nervous disease. The depressing passions, such as fright, alarm, disgust, terror, and rage have no doubt in all ages exerted a deleterious influence on the nervous system; but in the present day the keen competition evoked by the struggle for existence in the higher departments of social life must subject the latest evolved portion of the nervous system to a strain so great that only those possessing the best balanced and strongest nervous systems can escape unscathed.

## CHAPTER III.

## GENERAL SYMPTOMATOLOGY.

## GENERAL CLASSIFICATION OF ELEMENTARY SYMPTOMS.

THE nervous system, taken as a whole, is a mechanism by means of which all the actions of the individual are brought into relation with one another and with the actions of external agents upon the organism. Disease of the nervous system, therefore, is indicated objectively by a loss of harmony between the various actions of the individual, and consequently all the diseases of the nervous system may be regarded as a disturbance of the harmony of motor actions, or, in one word, as a motor inco-ordination. In a case of cutaneous and muscular anaesthesia of the upper extremity, for instance, the patient, unless guided by the sense of sight, will allow an object grasped in the hand to drop; or, in other words, there is a loss of co-ordination between the impressions made upon the surface and the muscular contractions by which a healthy organism responds. Nor is the case different when the absence of healthy reaction is due to paralysis of the muscles, since this also may be represented as a loss of co-ordination between afferent and efferent impulses. From this point of view all the diseases of the nervous system may be represented as disturbances of the motor functions or as *kinesioneuroses*. This generalisation is, however, somewhat too sweeping, since our objective knowledge of disease extends not simply to the actions of the organism under observation, but extends also to the changes of form induced by nutritive changes, including the alterations of secretion caused by disease of the nervous system. Our objective

knowledge, therefore, includes nutritive changes as well as inco-ordination of motor actions; or embraces *Trophoneuroses* as well as *Kinesineuroses*. So far we have spoken only of an *objective* knowledge, but our acquaintance with disease does not stop here, but also includes a *subjective* knowledge; and if the former can be resolved into a knowledge of motor and nutritive changes, the latter can be resolved entirely into a knowledge of disordered feeling. Each person can know both his own diseases, and the diseases of others, only through his feelings, and the inferences deduced from these feelings. And as all diseases may be resolved objectively into *Kinesineuroses* and *Trophoneuroses*, so all can be resolved subjectively into disorder of feeling or *Æsthesineuroses*. But instead of maintaining this double classification from two points of view, the two classifications are combined in practice; so that some symptoms, although made known to us by disordered actions, are rapidly translated by us into feeling and regarded subjectively; while other symptoms, even when only known through feeling, are rapidly translated objectively, and regarded as motor or nutritive disorders.

If, for instance, a slight touch on the cutaneous surface of the hand of a patient is followed by numerous contractions of the muscles of the extremity, causing the hand to be suddenly withdrawn, and also by contractions of the muscles of expiration and of vocalisation, inducing a loud cry, our immediate inference is not that this local impression was accompanied by an undue amount of motor reaction, but that it was accompanied by an undue amount of sensibility; the case is regarded not as an instance of the *Kinesineuroses* but of the *Æsthesineuroses*. Suppose, however, that the observer himself is the patient, and is suffering from double vision, and that he finds himself unable to direct his right eye to an object situated to his right without turning his head; if he is sufficiently informed he regards the case not primarily as one of sensory disturbance, although this is all he can know of it from direct knowledge, but as due to paralysis of the external rectus of the right eye; in short, he classifies the disease not amongst the *Æsthesineuroses* but the *Kinesineuroses*. It is manifest, therefore, that the distinction between the two



classes of disease is to a very considerable extent arbitrary, and that what may be regarded, from one point of view, as a disorder of sensibility may, from another point of view, be regarded as a disorder of motor actions. And if this be true with respect to the *Æsthesioneuroses* and the *Kinesioneuroses*, it is no less true with respect to the *Kinesioneuroses* and the *Trophoneuroses*. Some forms of paralysis, for instance, are closely associated with muscular wasting; so that both the motor disorder and the trophic change are but manifestations of one and the same affection. Certain paralyses of peripheral and spinal origin are associated with atrophy of the affected muscles, and indeed I might add of the affected nerves also, so that these affections could be included amongst the *Trophoneuroses* as well as the *Kinesioneuroses*. But however imperfect may be the classification which divides the diseases of the nervous system into sensory, motor, and nutritive disorders, its practical utility overrides all theoretical objections.

§ 38. *Æsthesioneuroses*.—A sensation is the conscious state or feeling resulting from the action of external things on some part of the body. Sensations have two mental functions to perform: the one intellectual and the other emotional, and they may consequently be subdivided into (1) intellectual and (2) emotional sensations.

(1) *Intellectual Sensations*.—In intellectual operations sensations are variously combined with each other and with representations of sensations previously experienced; so that the mind forms a judgment of the objective cause of the sensation; or, in other words, a perception of an external object is formed.

(2) *Emotional Sensations: Pleasures and Pains*.—In emotional states a sensation is regarded as agreeable or disagreeable, or as giving rise to pleasure or pain. This is not the place to advance a complete theory of pleasure or pain; but it may be remarked that an agreeable feeling is caused by a certain moderate exercise of every part of the sensory mechanism, and that a more or less diffused liberation of energy, throughout the whole or at least a large portion of the nervous system, is the physical condition of intense pleasure.

When, on the other hand, a considerable portion of the sensory mechanism is not duly exercised a feeling of discomfort is occasioned; and when there is a large liberation of energy in a limited portion of the sensory apparatus, in a short time, acute pain is occasioned. Excess of the normal stimulus of a peripheral end organ causes pain. A strong light or a loud noise also causes a painful feeling; although the feeling caused by excess of stimulus to the end organs of the cutaneous sensory nerves, or to the afferent nerves themselves, is the type to which we usually refer our elementary painful feelings.

(i.) *Relation of the strength of the Stimulus to the intensity of the resulting sensation in Health.*—The intensity of a sensation varies according to the strength of the external stimulus, and the amount of the irritability, and the consequent degree of the specific resistance of the sensory mechanism.

The sensory mechanism consists of—(1) a peripheral sense-organ for the reception of external impressions; (2) nerve paths for the centripetal conduction of these impressions; and (3) a central sense-organ for the reception of the conducted impressions. The degree of the irritability of the sensory mechanism varies greatly within physiological limits; but for practical purposes it may be regarded as constant in health, and the intensity of the sensation as varying only in proportion to the strength of the external excitant. The relation between the strength of the stimulus and the intensity of the resulting sensation is not, however, a simple one, even under healthy conditions.

A candle lighted in a dark room gives us a luminous sensation of a certain intensity; but a second lighted candle, although it increases, yet does not double the intensity of the sensation, and a third candle produces still less effect. The true relation between the degree of stimulus and the intensity of the sensation was first discovered by determining what increase, of a stimulus already in action, causes the smallest possible increase of sensation. Weber was the first to formulate the law of relation between the stimulus and the resulting sensation. Weber's law is: that the increase of stimulus necessary to produce the smallest possible increase of sensation is, within certain limits, directly proportional to the strength of stimulus already applied.



This law, which is applicable to all the organs of sense, has received a more definite enunciation from Fechner. Regarding sensation as the sum of a series of increments of sensation, corresponding to increments of stimulus, Fechner adopted the mathematical operation of integration, and concluded that sensations increase, not in proportion to the strength of the stimulus, but to the logarithm of the strength of the stimulus.

(ii.) *Relation of the strength of the Stimulus to the intensity of the resulting sensation in Disease.*—The intensity of a sensation varies in disease, not only according to the strength of the external stimulus applied, but also according to the degree of the irritability of the sensory mechanism itself. When the irritability of the cells and fibres which constitute the sensory mechanism is in any way altered, there is a corresponding change in the intensity of the sensation caused by an applied stimulus, the healthy reaction being, of course, regarded as the standard of comparison.

(1) *Hyperæsthesia.*—If the irritability of the cells and fibres which constitute the sensory mechanism is increased, the intensity of the sensation resulting from an applied stimulus is also increased in a corresponding degree. When a stimulus, such as a touch on the skin, which in health gives rise to a very slight degree of an intellectual sensation, either causes an undue amount of the same, or, what is more usual, induces a painful feeling, the condition is called *Hyperæsthesia*.

(2) *Hyperalgesia.*—When a stimulus, such as the prick of a pin, which in health causes a slight degree of a painful feeling, causes an undue amount of the same feeling, the condition is called *Hyperalgesia* or *Hyperalgia*.

(3) *Anæsthesia.*—When the irritability of the cells and fibres constituting the sensory mechanism is diminished or abolished, the sensation resulting from a particular stimulus becomes also diminished or abolished. When the intellectual sensations are diminished or abolished, the condition is called *Anæsthesia*. The term *Hypæsthesia* has been introduced by Eulenburg to indicate diminution of sensory reaction, while he limits the meaning of *anæsthesia* to its abolition. But, in order not to multiply words, the latter term will be used in the subsequent pages in a general sense as including both conditions.



(4) *Analgesia*.—When, on the other hand, the painful feelings induced by the application of stimuli are diminished or abolished, the condition is called *Analgesia* or *Analgia*. Eulenburg has proposed the term *hypalgesia* or *hypalgia*, to indicate diminution of painful reaction, while limiting *analgesia* to its abolition. But we prefer to dispense with this refinement.

(5) *Neuralgia*.—Pain is often a symptom of disease in the absence of all external stimulation. Severe pain accompanies local inflammation and other diseases, but at times severe paroxysms of pain, limited to the course of a sensory nerve, is the prominent symptom of the disease, and when this is the case the condition is called *Neuralgia*.

(6) *Paræsthesia*.—Various other elementary disorders of sensation are experienced, independently of any external impressions, but which cannot be included under any of the previous categories, and are therefore grouped under the general term *paræsthesia*. The cutaneous paræsthesiæ are such sensations as pruritus, tickling, numbness, furriness, crawling, feelings of heat and cold. The visceral paræsthesiæ, which form a very important group of symptoms, are such feelings as hunger, thirst, nausea, disgust, the feelings of suffocation and fainting, and other organic sensations. In so far as these feelings are painful they may be called *Paralgesia*.

(iii.) *Retardation and Acceleration of Sensory Conduction*. The interval which elapses between the instant at which a stimulus is applied to a sensory surface and the moment at which the subject makes a voluntary signal to indicate that the sensation has been perceived, has been called by Exner "the reaction period." The normal "reaction period" may be either increased or diminished. The conduction of centripetal impressions is accelerated in cases of hyperæsthesia, and retarded in cases of anæsthesia; hence the length of the reaction period is diminished in the former, and increased in the latter. Retardation of conduction, however, is a much more important and reliable evidence of disease than acceleration, inasmuch as the former is much more easily estimated than the latter. The normal reaction period is indeed so short that acceleration of conduction can only be detected by the aid of

refined physiological mechanism ; but retardation of it, on the other hand, may occur to such an extent as to be readily appreciable to the unaided senses, and the symptom thus affords a valuable aid in diagnosis.

§ 39. *Kinesioneuroses*.—Muscular contraction is caused both in health and disease by irritation of some part of the nervous system. The irritation may have its seat in the intra-muscular nerve endings, the end plates of the striated muscles, or the terminal fibrils of the unstriated muscles ; it may have its seat in the cells of the peripheral ganglia, spinal cord, or brain ; or the contraction may occur indirectly from irritation of the centripetal nervous apparatus.

All movements may be subdivided into three classes, viz., the voluntary, the reflex, and the automatic.

(1) *Voluntary Movements*.—Voluntary movements are caused by stimulation of circumscribed regions of the cortex of the brain, called psycho-motor centres, the impulses being conducted downwards by the fibres of the *pyramidal tract* to the spinal cord, and through the motor nerves to the muscles.

(2) *Reflex Movements*.—Simple reflex movements are caused by stimulation of afferent fibres or of their peripheral terminations, the impulses being conveyed either to peripheral ganglia or the grey substance of the central grey tube, and reflected by the latter through the efferent fibres to the muscles.

(3) *Automatic Movements*.—The automatic movements are those which occur spontaneously, unconsciously, and in the absence of external irritation. To this class belong the contractions which are concerned in maintaining various attitudes in space, and which are probably co-ordinated in the cerebellum, as well as movements indicative of various instincts, probably co-ordinated in the basal ganglia. But although the definition of automatic movements implies the absence of peripheral stimulation in their genesis, it is quite certain that many of the movements which are usually regarded as automatic are induced by external stimulation. It is indeed exceedingly difficult to say when a reflex movement ends and a voluntary or an automatic movement begins. Practically, however, the distinctions between these three classes of movements are valid, and



the theoretical difficulties in defining them need not be discussed further at present.

§ 40. *Relation of the strength of the Stimulus and the resulting Contraction.*—But whatever may be the nature of the resulting movements, the muscular contractions bear a certain quantitative relation to the liberating stimulant. The quantity of motor innervation which reaches a muscle depends upon two factors, the primary irritant and the irritability of the nervous apparatus; and when the latter is constant the strength of the contraction is proportional to the strength of the primary irritant. The degree of irritability of the nervous system, as a whole and in its various parts, varies to a considerable extent within physiological limits; but the regularity of our voluntary movements, and of the respiratory and cardiac rhythm, and of other involuntary movements, depends upon the constancy of the degree of irritability of the various parts of the nervous system; so that in health the strength of the contraction and of the primary irritation are proportionate to one another. In diseased processes, on the other hand, the irritability of the various sections of the nervous system is greatly altered, so that no assignable proportion exists any longer between the irritation and the resulting contraction.

§ 41. *Varieties of Elementary Motor Disturbances.*—When the irritability of the nervous motor mechanism becomes in any way altered it gives rise to a disproportion between the strength of an applied stimulus and the resulting muscular contraction, a disproportion which may declare itself by excess or diminution of reaction or by abolition of all muscular contraction.

(1) *Hyperkinesis.*—When a stimulus gives rise to muscular contractions which are in excess of the normal proportion, or when an undue muscular contraction occurs in the absence of all recognisable external stimulation, the condition is called *hyperkinesis*.

(2) *Akinesis.*—When there is a diminution or abolition of the power of exciting the muscles to contraction, either by voluntary, reflex, or automatic excitation, the condition is called *akinesis*. The term *hypokinesis* has been introduced by



Eulenburg to designate the diminution of motor reaction to excitation, while he limits akinesis to its abolition; but, in order not to multiply words, the latter term is used here in a generic sense as including both conditions.

It is doubtful whether qualitative changes of motor activity are ever observed; hence it is unnecessary to form a third group of motor disturbances termed *parakinesis*, corresponding to the sensory affections termed *paræsthesia*.

(3) *Synkinesis*.—Although motor affections do not present any appreciable qualitative alterations, yet various motor anomalies are frequently observed in connection with paralysis or spasm of some of the muscles of groups associated in their actions, and these may be included under the term *Synkinesis*.

§ 42. *Trophoneuroses*.—Various experimental and pathological facts are now accumulated which have led physiologists to assume the existence of trophic fibres in the peripheral nerves. The nerves which preside over nutritive functions are of two kinds. (1) Trophic nerves, in the narrow sense, disease of which induces nutritive disorders in various parts of the body; and (2) Secretory nerves, disease of which induces, as a concomitant of nutritive changes in the glands to which they are supplied, both quantitative and qualitative changes in the secretion of these glands. That the secretory nerves are not identical with the vaso-motor nerves, as was at first conjectured, has been proved by the experiments of Ludwig, V. Wittich, and Heidenhain, with respect to the secretion of the parotid and submaxillary glands. Ludwig showed that the secretion of saliva is independent of the arterial pressure, since irritation of the nerves of the salivary glands causes the manometric pressure to be greater in Wharton's duct than the arterial pressure in the carotid, and the temperature within the gland also to rise above that of arterial blood. V. Wittich also proved that irritation of the cervical sympathetic produces similar phenomena in the gland, even after it has been rendered anæmic by the carotid having been previously tied. Heidenhain again proved with respect to the submaxillary gland that certain poisons, such as sulphate of atropine, paralyse the secretory nerves, while leaving the

circulation of the gland completely undisturbed. If a canula be placed in Wharton's duct in an atropised and curarised animal and the chorda tympani be irritated, not a drop of saliva will flow, even if the action on the vessels of the gland be normal.

The elementary nervous nutritive affections may be subdivided into those which are characterised by excess or by diminution of nutritive activity, manifested by an increase or diminution in the volume, consistence, or colour of a certain tissue or organ, or in an increase or diminution of the secretion of certain glands.

(1) *Neurotic Hypertrophy*.—Excess of nutritive activity does not appear always to depend upon an increase of the functions of the trophic nerves. Both irritation and complete division of the trophic nerves may be followed by proliferation of the elements of the tissues and increased size of organs, so that apparently identical causes may give rise at one time to hyperplastic processes and enlargement of organs, and at another to hypoplastic processes and atrophy of organs. Mantegazza observed hypertrophy of the connective tissue, and of the periosteum, bones, and lymphatic glands after experimental section of nerves in animals. And, similarly, division of the large nerve-trunks in man, especially those of the extremities, is not unfrequently followed by circumscribed hyperplastic phenomena in certain parts, as the skin, nails, hairs, bones, and joints, side by side with atrophy of other parts, as of the muscles and subcutaneous tissue. Similar phenomena are also observed in destructive processes in some of the nerve centres. The experiments of Claude Bernard and others show that increase of the salivary secretion may occur after section of the cervical sympathetic, and therefore independently of its influence. It is probable that in such cases we may have to do with nerves which either directly or by reflex action exert a regulating or moderating influence upon the nutrition and secretion of the parts to which they are distributed, and section of which removes this regulating or inhibitory influence. Other hyperplastic phenomena, such as the interstitial development of fat in pseudo-hypertrophy of the muscles, depend probably upon chronic diseased irritative processes in the central nerve cells.



(2) *Neurotic Atrophy* consists of those processes which, in consequence of an abnormal trophic innervation, are accompanied by diminution of the volume, colour, and consistence of certain tissues and organs. Certain forms of arrest of development and growth, and of degenerations, along with certain forms of abnormal diminution of secretion, belong to this category. The diminution of secretion may be brought about through a paralysis of the acceleratory, or irritation of the regulatory or inhibitory nerves of secretion. Diminished nutrition of parts is probably induced by irritation of trophic fibres. Neuralgia is frequently associated with diminished nutrition of the parts to which the affected nerve is distributed. In trigeminal neuralgia, for instance, there is frequent loss of the colour of the hair on the affected side of the head, or of the cilia and supercilia, as well as atrophy of the skin and of the remaining soft parts, and also of the bones of the face. From what has just been said it would appear that irritation of the trophic and secretory nerves is at one time followed by hyperplasia and increase of secretion, while at another time it is followed by the opposite conditions of atrophy and diminished secretion. Division of these nerves, on the other hand, is also followed by the same phenomena of excess at one time and diminished nutrition at another. It is not improbable, therefore, that there are two kinds of trophic and secretory nerves, the one acting as accelerators and the other as regulators of nutrition corresponding to the acceleratory and inhibitory nerves of the heart.

(3) *Neurotic Paratrophies and Dystrophies*.—Besides the conditions of abnormal increase and diminution, qualitative alterations of nutrition are observed, and these may be called *paratrophies* or *dystrophies*. It is not improbable that the nervous system is often implicated in the growth and development of new formations. Qualitative changes of nutrition occur in consequence of abnormal nervous influences and also of certain conditions which are usually regarded as neuroparalytic in their character. The arthropathies of tabes dorsalis, the tenosynitis of apoplectic hemiplegia, the circumscribed erysipelas and glossy fingers which occur after injury to the peripheral nerves, are trophic disturbances which cannot be regarded either as hypertrophies or atrophies. It is difficult in those cases to estimate



how much is due to disturbances of the vaso-motor and of the trophic nerves. But it is probable that herpes and various cutaneous eruptions are caused by disease of trophic cutaneous nerves, or by an ascending neuritis extending to the ganglia of the posterior roots. The most probable explanation of the affection of the eye which has been described under the name of neuroparalytic ophthalmia is that it is caused by injury of trophic fibres which descend from the Gasserion ganglion or which are at least found in the trunk of the trigeminus. But another supposition may be made. It may be supposed that during health the sensory nerves of a part of the body exercise a continuous influence on the trophic nerves of the same region, similar to the nervous influence which maintains the vascular tonus. The intervertebral and the trigeminal ganglia, and probably also nerve centres situated higher in the cerebro-spinal system, may be regarded as part of this reflex mechanism of nutritive tonus. Abnormal increase or diminution of this healthy excitement in the sphere of the sensory nerves may lead to nutritive affections which assume, according to circumstances, the form of hypertrophy, atrophy, or paratrophy. These trophic disturbances may therefore be called reflex trophoneuroses, and they may be subdivided into those of an irritative or depressive character. This distinction, although theoretically tenable, is, in the present state of our knowledge, incapable of being practically applied, inasmuch as it is impossible to discriminate the reflex trophic disturbances from those which are of direct origin. It is possible, however, to maintain the distinction in the case of disturbances of secretion.

Elementary nervous symptoms may also be classified according to the tissues and organs affected. This anatomical subdivision cuts across at right angles the physiological one just described, so that both may be combined in the following scheme:—

I.—*Esthesioneuroses*.—

- (I.) Sensory affections of the skin, including the external and exposed portions of the mucous membranes.
- (II.) Sensory affections of the voluntary muscles.

- (III.) Sensory affections of the passive parts of the locomotive apparatus as the bones and joints.
- (IV.) Sensory affections of the internal parenchymatous organs.
- (V.) Sensory disturbances of the special senses.

These groups may be briefly characterised as cutaneous, muscular, articular, visceral, and sensory *Æsthesioneuroses*.

## II.—*Kinesioneuroses*.—

- (I.) Motor affections of the muscles of external relation or of the striped muscles, or *external kinesioneuroses*.
- (II.) Motor affections of the internal organs or *visceral kinesioneuroses*.
- (III.) Vaso-motor disturbances—*vascular kinesioneuroses*, or *angioneuroses*.

## III.—*Trophoneuroses*.—

- (I.) Trophic affections of the nervous system itself.
- (II.) Trophic changes of the voluntary muscles.
- (III.) Trophic affections of the skin and epidermoidal structures.
- (IV.) Trophic affections of the joints and bones.
- (V.) Nutritive and secretory disturbances of the glandular apparatus.
- (VI.) Trophic affections of the internal organs.

## CHAPTER IV.

## I.—ELEMENTARY AFFECTIONS OF INDIVIDUAL SENSORY MECHANISMS.

## (I).—SENSORY DISTURBANCES OF THE SKIN (CUTANEOUS ÆSTHESIONEUROSES).

§ 43. *General and Special Cutaneous Sensations.*—The sensations which are communicated through the skin may be divided into *general or common sensations* and *special or tactile sensations*; and each of these groups may be divided into several varieties. The first group consists of such sensations as pain, itching, titillation, sensual pleasure, and that resulting from electrical excitation. The second group consists of the specific sensations of pressure and temperature, which arise in response to mechanical and thermal stimuli. Recent observations appear to show that painful irritations of the skin are due not to excessive irritation of the ordinary nerve terminations, but to irritation of special end-organs.

§ 44. *Tactile and Pathic Channels.*—There seem to be different channels of conduction in the spinal cord and brain for tactile and painful sensations, called respectively *tactile* and *pathic* channels. It does not appear improbable therefore that these channels are connected with different peripheral terminal organs. In disease one or more of these channels may be disturbed while the others remain unaffected; so that pathological conditions may be said to analyse the various sensations into their elementary forms. In certain diseases of the brain or of the spinal cord hyperæsthesia as regards temperature has been observed while sensitiveness to pressure was unaffected; and, conversely, cases have been described where the



patient could tell when he was touched, but could not distinguish between hot and cold bodies. An analogous analysis of the sensations of temperature and of pain may be effected by a simple experiment. If the elbow is dipped into a very cold fluid, the cold is only felt at the immersed part of the body; while pain is felt in the finger points supplied by the ulnar nerve.

### § 45. *Methods of Examining the Sensory Apparatus*

Inasmuch as sensory affections can only be known through the statements and gestures of the patient it is necessary to proceed systematically with our investigation of them, and to employ various methods of testing the sensations of our patients so that the conclusions derived from one method may be checked, and corrected or confirmed by the inferences drawn according to other and different methods.

1. *Test of Common Sensations.*—Cutaneous common sensations may be tested by the prick of a pin, by burning, pinching, and firm pressure. The most certain method, however, is the application of the faradic current; since it can be carefully graduated so as to determine the minimum stimulus which causes either a common sensation or a sensation of pain.

2. *Test of Rapidity of Sensory Conduction.*—It is sometimes very useful to determine the period which intervenes between the application of a stimulus and the resulting sensation, inasmuch as we have seen that conduction of centripetal impressions is retarded in cases of anæsthesia, and accelerated in cases of hyperæsthesia.

The following are the general results obtained by physiologists for healthy individuals (after Mc.Kendrick):—

Nature of Stimulus.	Time between application of stimulus and signal of perception, in fractions of a second.	Name of Observer.
Shock on left hand .....	·12	Exner.
Shock on forehead .....	·13	Do.
Shock on toe of left foot .....	·17	Do.
Sudden noise .....	·13	Do.
Visual impression of electric spark.	·15	Do.
Hearing a sound .....	·16	Donders.
Current to tongue causing taste...	·16	Von Wittich.
Saline tastes .....	·15	Vurtschgau & Hönigschmid.
Taste of sugar .....	·16	Do.
Taste of acid .....	·16	Do.
Taste of quinine .....	·23	Do.

Any serious departure from the figures given in this scale, either by way of acceleration or retardation would indicate disease. Acceleration of the conduction cannot however be recognised without the use of delicate instruments; hence it is only necessary for practical purposes to take the retardation into account.

3. *Tests of Sense of Pressure.*—When a body is in contact with any part of our skin we are conscious, not simply of a sensation of pressure, but also form a judgment with respect to the part of our body which has been touched, and the nature of the substance touching it. By sensations of pressure we are able to come to the following conclusions: (1) We infer the existence of some substance touching our body; (2) Variations in the intensity of the pressure exerted helps us to estimate the weight of the touching body; (3) The cutaneous surface forms a tactile field similar to the field of vision, which enables us to localise our sensations of touch and to refer them to definite parts of the surface of our bodies; (4) Closely connected with our power of estimating weight from sensations of pressure is the power of discriminating between two or more cutaneous points simultaneously touched.

The sense of pressure must be tested by the method introduced by H. Weber. This method consists of the super-imposition of weights in order to determine the smallest difference which can be perceived. The testing may be carried on by ounce, half-ounce, and drachm weights, or by various coins. The variations in the sensations bear that general relation to the variations in the super-imposed weights which is formulated in Fechner's law. In order that the muscular sense may be excluded, it is necessary that the part of the body to be investigated should rest on a firm support, and the sensation of temperature must be excluded by the interposition of a bad conductor of heat, such as a small wooden disc.

Eulenburg has constructed an instrument for testing the sense of pressure, which he has named *baræsthesiometer*. It consists essentially of a spring balance, constructed so that varying degrees of pressure on the spring may be read off on a dial. By means of this instrument Eulenburg found that a differential pressure of  $\frac{1}{16}$  to  $\frac{1}{8}$  in. can be discriminated by the skin of the face—the skin of the forehead being especially sensitive. The perception of difference amounted to from  $\frac{1}{16}$  to  $\frac{1}{8}$  in. on the skin of the hand and arm, and on the anterior aspect of the thigh and leg, and on the dorsum of the foot; but is much less acute on the skin of the sole of the foot and back of the leg. When the affection is unilateral, so that one side can be compared with the other, the results obtained are much more reliable than when both are affected, inasmuch as in the latter case a standard of comparison must be found in another subject. It may be mentioned that a colder weight is felt to be heavier than one of the same temperature, or as slightly warmer than the body; so that the apparent difference of pressure between two bodies becomes greater when the heavier weight is the colder, and less when the lighter weight is the colder of the two. The sensitiveness is



also greater for an increase than for a decrease of the difference in the weights, and also greater for a small absolute than for a large absolute pressure.

Goltz has introduced a very efficient method for determining the smallest possible variation in pressure, affecting a cutaneous part, which can be perceived. It consists of an india-rubber tube filled with water, and by rhythmical pressure waves analogous to those of the arterial pulse are produced. In order to secure a constant surface of contact the tube is bent over a piece of cork at the spot which is intended to be in contact with the cutaneous part to be examined. This method gives the same scale of results as the experiments of Weber with the compasses, to be mentioned immediately, with the exception of the tip of the tongue, in which the sense of pressure, as determined by Goltz's method, stands much lower than in Weber's scale for measuring the sense of locality.

4. *Tests of the Sense of Locality.*—The sense of locality may be tested by touching some part of the skin with the finger or point of a needle whilst the patient's eyes are closed, and requesting him to indicate the point touched. According to E. H. Weber the error in healthy persons is about equal to the minimum distance at which the points of a pair of compasses can be recognised as separate in different parts of the skin. The distance at which the points of the compasses must be separated from one another, in order to be felt as two, varies for different regions of the body. The following may be taken as the normal scale, from which any marked deviations must be regarded as pathological:—The tip of the tongue, 1·18 mm.; the extremities of the fingers, 2·25 mm.; the dorsal side of the first phalanx, 16 mm.; the back of the hand, 31 mm.; the upper arm and thigh, 77 mm. The smallest required distance is less in the transverse than in the longitudinal direction in the limbs; it is less when the points are placed on the skin one *after* the other; it is less when the surfaces vary in structure, as the inner and outer surface of the lips; it is less when beginning with larger distances and gradually diminishing the distance until both sensations blend, than when beginning with the smaller distances and gradually finding that distance at which the two separate sensations make their appearance; and it also becomes smaller by practice. The power of discrimination is greater when the points of the compasses are moved instead of being kept stationary; hence in testing the sense of locality it is useful to move the points of the compasses over the skin in two parallel lines, and to note the variations in the apparent distance which occurs in different localities. The compasses will seem to the patient to widen in the most sensitive parts, and to narrow or blend in the less sensitive parts. The points of the compasses may be blunted with sealing-wax; or points of some non-conducting substance, such as ivory or wood, may be used so as to eliminate the effects of the sense of temperature. It may be mentioned that when the points of the compasses are just sufficiently separated to give rise to two distinct impressions, if one of the points



be pressed forcibly, the other ceases to be distinguished, and the two impressions blend when the skin between the points is irritated by tickling or by induction currents.

In order to explain these phenomena it is necessary to assume that the surface of the skin forms a field of tactile sensibility which is composed of tactile areas or units. The tactile sensation is a symbol to us of some external event, and we refer the sensation to its appropriate place in the field of touch. The spot to which the sensation is referred, however, does not correspond accurately with the spot irritated, but to the surface of variable extent surrounding this point and which constitutes the tactile area or unit. When two such areas, which partly touch or overlap each other, are stimulated, they cannot be separately perceived, and the separation does not take place until there is a non-irritated sensory portion between the two stimulated areas. These tactile areas, however, cannot be regarded as fixed and as corresponding to the anatomical distribution of the terminal nerve-fibres, since such an area exists round each cutaneous point; and the improvement by attention and practice in the sense of touch cannot be supposed to be due to the growth of new nerve-fibres in the skin. It must, therefore, be assumed that the transmission of the irritation of one nerve-fibre to another nerve-fibre in the neighbourhood is a central process. It may be conjectured that there are central sensation areas corresponding to the peripheral ones; and that it is by a more exact limitation of these areas the improvement in the sense of locality takes place.

When the parts specially used for discriminating the finer distinctions between objects, as the tips of the fingers and the palms of the hands, are to be tested, the patient may be allowed to handle small common objects, as pieces of money or buttons, and asked to describe them with closed eyes. It may also be ascertained whether the patient can distinguish by touch alone a smooth and a rough surface, or a hard and soft object, or whether he can with closed eyes distinguish the head from the point of a needle.

5. *Tests of Sensations of Temperature.*—When the temperature of the skin is raised or lowered we experience sensations of heat and cold respectively; and when the variations of temperature are produced by bodies in contact with the skin we form judgments with respect to their temperature. The range of most accurate sensation lies between  $27^{\circ}$  and  $33^{\circ}\text{C}$ , then variations between  $33^{\circ}$  and  $39^{\circ}\text{C}$ , and lastly between  $14^{\circ}$  and  $27^{\circ}\text{C}$  (Nothnagel). Variations above and below these limits are no longer specifically felt, but produce general sensations of feeling rapidly rising to pain. The change in temperature is felt more intensely the more rapid its occurrence, and the larger the affected surfaces.

The sense of temperature may be roughly tested by the application of hot and cold bodies; but various means have been adopted to ensure greater accuracy and delicacy. A ready and delicate test is to see whether the patient can distinguish with certainty between a cool current

of air, such as may be produced by blowing upon the part from a distance, and a warm one produced by breathing upon it in close proximity. Another method is to put the part successively into water of various temperatures, so as to determine the smallest difference of temperature which can be perceived. Weber employed for this purpose small phials filled with oil of known temperature. In practice it is more convenient to use two test tubes, one filled with cold and the other with warm water. Both Nothnagel and Eulenburg have constructed *thermæsthesiometers* for this purpose. Nothnagel found the smallest perceptible difference of temperature to be the following :—On the breast,  $0.4^{\circ}$ ; on the back,  $0.9^{\circ}$ ; back of the hand,  $0.3^{\circ}$ ; palm of the hand,  $0.4^{\circ}$ ; arm,  $0.2^{\circ}$ ; back of the foot,  $0.4^{\circ}$ ; lower extremity from  $0.5^{\circ}$ — $0.6^{\circ}$ ; cheek,  $0.4^{\circ}$ — $0.2^{\circ}$ ; the temples,  $0.4^{\circ}$ — $0.3^{\circ}$ . These results conform in the main with what had previously been ascertained by Weber.

#### § 46. *Cutaneous Anæsthesia.*

Cutaneous Anæsthesia is characterised by diminution or abolition of the function of the cutaneous sensory mechanism. When the sensibility to pain is diminished or abolished the condition is designated *Analgesia* or *Analgia*, and Eulenburg has proposed to call the condition in which tactile sensibility is alone affected by the name of *Apselaphesia* (Eulenburg).

Diminution of tactile sensibility is not necessarily associated with a corresponding condition of common sensibility. Diminution or abolition of tactile sensibility may indeed be associated with cutaneous hyperalgesia; and, conversely, cutaneous analgesia may be associated with increased acuteness of tactile sensibility. And even the various forms of tactile sensibility, or the senses of pressure, of temperature, and of locality, are not always equally affected, since one or more of them may be weakened or abolished while the others remain unaffected or even increased.

*Total and Partial Sensory Paralysis.*—If all forms of cutaneous sensibility are lost, then the condition is called *total sensory paralysis*; but if the tactile sensations are diminished or abolished while the common sensations are preserved, or, conversely, if the common are lost while the tactile sensations are preserved, the condition is called *partial sensory paralysis*. And, again, if one or more of the tactile or common sensations are weakened or lost while others are preserved, the conditions are called respectively *partial tactile paralysis* and *partial*



*paralysis of common sensation.* All possible combinations of partial paralysis of the various forms of sensation have been observed and described; but the most usual combination observed is the loss of the sensation of pain (analgesia), with complete preservation of the tactile sensibility.

§ 47. *Symptoms.*—Diminution or loss of cutaneous sensibility declares itself by the statements and gestures of the patient. The statements of the patient are often very vague, and can only be relied upon when they are made in answer to the definite tests which have already been described. These tests must be methodically applied, in order to determine whether every form of cutaneous sensibility is affected, or whether one or more of the forms of sensation are alone implicated. The extent and degree of anæsthesia must also be determined. Diminution or loss of tactile sensibility must be determined by applying the tests for the senses of pressure, temperature, and locality; while diminution or loss of the common sensations must be determined by pinching, tickling, application of the faradic brush, and pricking with a needle.

In the higher degrees of anæsthesia the patient feels as if his limbs were altogether absent, and as if in walking he stepped on air. The usual sensations are wanting during manual labour, and small objects fall out of the hands unless the patient controls their action by means of the sense of sight.

*Associated Symptoms.*—The symptoms of anæsthesia arise partly from the diminution or abolition of normal sensations and partly from associated conditions. When cutaneous sensibility is diminished the patient frequently complains of various paræsthesiæ, as "numbness," "furriness," "crawling," or formication. The patient feels at times as if his fingers were covered with gloves, as if something were between the skin and the bodies touching it. During walking the patient may feel as if he were stepping on wool or on a carpet or on bladders filled with water.

Pain is frequently associated with anæsthesia when the affection is caused by injury to the peripheral nerves. The irritation which causes the pain is situated in a part of the sensory nerve above the point where its conduction is inter-



rupted; but, in accordance with the law of *eccentric projection*, the pain is referred to the surface. When severe pain has been felt in the anæsthetic part the condition has been called *anæsthesia dolorosa*. It is sometimes difficult to determine whether a particular case is to be regarded as one of anæsthesia neuralgia, or motor paralysis, inasmuch as the symptoms characteristic of each affection are combined in various ways. The special senses are sometimes affected, especially when the cause of the anæsthesia is central, or when the disease is situated in nerves which contain fibres of special sense, as the lingualis. Loss of taste and of smell are the most common affections of special sense which are associated with anæsthesia; loss of hearing is less frequent, and loss of sight is rare.

#### § 48. *Distribution of Anæsthesia.*

(1) *Circumscribed Anæsthesia*.—In this form the sensory paralysis is limited to the district supplied by certain nerve trunks, or it may even be limited to small circumscribed patches of the skin. The lesions which give rise to this form may be situated in the course of the nerves, their peripheral terminations, or posterior roots.

(2) *Anæsthesia in the form of a Girdle*.—In this form of the affection a zone of varying width surrounding the body on one or both sides is found to be anæsthetic. This zone may be situated at various levels, and may therefore pass round the pelvis, abdomen, thorax, or even the region of the shoulder or neck. It is caused by disease of the spinal cord or of its membranes having only a limited longitudinal extent, and implicating the posterior roots of the nerves or the posterior grey cornua.

(3) *Para-anæsthesia*.—At times the anæsthesia is limited to the lower extremities and lower half of the body, and the affection may then be called *paranæsthesia*. The affection may at times be unilateral or limited to one extremity or portion of the trunk. This variety of disease is usually caused by diseases of the spinal cord, which destroy a portion of it more or less completely in a transverse direction, and thus prevent afferent impulse being conveyed to the cerebrum; but it is sometimes of functional origin, and then is associated with hysteria.

(4) *Hemi-anæsthesia*.—The anæsthesia may be distributed over the entire half of the body, face and extremities included, the anæsthetic parts being usually separated from the healthy parts by the medium line. In these cases the special senses, as well as the accessible mucous membranes, are usually implicated in the affection, taste and smell being usually abolished on the anæsthetic side, but the acuity of hearing and vision on that side is only diminished, not abolished. In this variety of the affection the lesion is usually situated in the posterior part of the optic thalamus, and interfering with conduction through the fibres of the posterior third of the internal capsule, the optic radiations of Gratiolet. This variety is also often of functional origin, and is then associated with hysteria.

#### § 49. *Varieties of Partial Sensory Paralysis.*

(1) *Analgesia*.—The most usual form of partial sensory paralysis is that in which the sense of pain is abolished while the other forms of sensibility are either unaffected, or only affected to a slight extent. Analgesia may be circumscribed or distributed over the lower half of the body, a condition which may be called *para-analgesia*, or implicate the lateral half, constituting *hemi-analgesia*.

(2) *Thermo-Anæsthesia*.—Insensibility to heat or cold may at times occur as an isolated affection. When the sense of temperature, however, is the only one affected, its loss is usually limited to more or less circumscribed portions of the skin, as that over the backs of the feet and patches of the cutaneous surface of the leg.

(3) *Tactile-Anæsthesia*.—When there is loss of tactile sense without any other affection of sensibility, the anæsthesia is generally found distributed in patches over the skin of the lower extremities. The degree of tactile sensibility must be estimated by a methodical application of the tests already described. Of all the diseases which give rise to partial forms of paralysis, locomotor ataxy is the most common and important. In this affection the posterior roots of the nerves are diseased; but the disease being chronic and progressive the root-fibres are implicated gradually and successively, and not simultaneously.



(4) *Retardation of Conduction in Partial Sensory Paralysis.*—Retardation of sensory conduction may be employed as a test of any form of anaesthesia, but in the partial varieties it gives rise to several anomalous phenomena which demand separate mention.

(a) *Separation of Tactile and Painful Impressions.*—Remak, Leyden, and several other authors have drawn attention to the fact that in cases of locomotor ataxy the prick of a needle causes a prompt feeling of touch, which is frequently followed in two or three seconds by a feeling of pain. In cases of thermo-anaesthesia, a test tube holding hot water may at first give rise to an immediate feeling of touch, to be followed in two or three seconds by a sensation of temperature.

(b) *Double Painful Sensations.*—Another curious sensory phenomenon has been described by Naunyn. He noticed in several cases of locomotor ataxy that pricking the skin on the back of the foot with a needle was followed by a first painful sensation, and when this had subsided by a second painful sensation, the latter being usually of greater intensity and longer duration than the former. The first is felt after a lapse of two and a half to three seconds subsequent to the prick, but the second is not felt until after another interval of from two to five seconds. This author found that the patients did not always experience a distinct interval between the two sensations, and this blending of two sensations which are occasionally distinct leads us to mention one or two other anomalies which are probably due to retarded conduction, and are at least found under the same physical conditions.

(c) *Persistent after-Sensations.*—The first of these consists of very persistent after-sensations. When the skin of the patient is pinched or pricked with a needle the resulting sensation begins more slowly, gradually increases in intensity, is more severe, and much slower in its disappearance on the diseased than on the healthy side. The fact that the sensation begins more promptly on the sound than on the diseased side shows that, in the case of the latter, sensory conduction is retarded. But retardation of conduction means increase of the specific resistance of the afferent conducting apparatus, which, in its turn, means that the impulses which reach the cortex of the brain in the



diseased must be stronger than on the healthy side in order to overcome that resistance, and, being stronger, they give rise to the stronger and more persistent pain.

(d) *Inability to Count Successive Impressions*.—Closely connected with these persistent after-sensations is the inability of the patient to count correctly several impressions made in quick succession. Enumeration of successive impressions presupposes an interval to elapse between the sensation caused by each, but when the conduction is retarded each sensation is unusually prolonged so that the one does not fade before the other begins and counting becomes impossible. Schiff has shown, experimentally, that a transverse narrowing of the grey substance causes a retardation of the conduction of sensation, the degree of which corresponds closely with the amount of the grey substance destroyed. It may, therefore, be presumed that wherever retarded sensation exists there is disease of the posterior grey cornua of the spinal cord; a supposition which also explains why the retardation affects by preference the sense of pain.

#### § 50. *Cutaneous Hyperæsthesia.*

*Increase of the Tactile Sensibility (Hyperpselaphesia)*. Abnormal acuteness of tactile sensibility—to which Eulenburg has given the name of *Hyperpselaphesia*—declares itself by excessive reaction to the various methods already described for testing the senses of pressure and locality. The sensibility to minimum differences of pressure may be excessive, so that a much smaller difference than usual is perceptible. The diameters of the areas of sensibility may also be unusually small. Increase of the sense of locality is sometimes observed in acute cutaneous affections, such as in vesication, erysipelas, and zoster; and it may or may not be associated with a corresponding increase of the sense of temperature, and of the cutaneous common feelings.

*Polyæsthesia*.—Brown-Séquard first drew attention to the fact that under some circumstances one point of the compass is felt by the patient, on being placed on the skin, as two, three, or five points. Fischer proposes to call this condition *Polyæsthesia*.

Local hyperæsthesia of the tactile sensibility may be induced experimentally. On passing a moderate constant current through the skin, increase of the sense of locality may be found at the cathode (Suslowa). When local anæmia is artificially produced by an extremity being maintained in an elevated position, the sense of temperature is increased, while there is a diminution of the sense of locality in the affected portions of skin. The tactile sensibility is also increased by carbonic acid baths, and by chloride of sodium and sea baths.

It is very probable that many cases of nervous palpitations and of the sensation of pulsation in many of the arteries of the body are due to a hyperæsthesia of the sense of pressure.

§ 51. *Distribution of Hyperæsthesia.*—The distribution of hyperæsthesia is more or less similar to that of anæsthesia. It may occur in more or less circumscribed patches, or be limited to the distribution of individual nerves, or it may implicate the lower half or the lateral half of the body, just as has already been described with respect to anæsthesia. Hyperæsthesia frequently precedes anæsthesia; and, as previously observed, some forms of sensibility may be in excess, while others are greatly diminished.

*Girdle Sensation.*—A subjective perception which produces the impression of having a girdle or a broad bandage tied about the trunk or limbs is a very common accompaniment of all spinal diseases. When situated at the upper part of the thorax, it may be accompanied with a severe sense of pressure, and is always very troublesome to the patient. It may occupy various levels on the trunk, but may also attack various parts of the lower extremities, particularly in the region of the ankle and knee, of one or both sides. The sensation is probably produced by a slight excitation of the entering posterior roots in cases when the spinal affection is limited in its longitudinal extent. It usually accompanies inflammatory or other morbid conditions of the cord, and originates with the root-fibres which occupy the upper limit of the disease. Any sort of local disease of the cord and its neighbouring parts which irritates the posterior roots to a moderate extent may produce the symptom.

§ 52. *Cutaneous Thermo-Hyperæsthesia.*—The sensibility to minimum differences of temperature may be in excess. This occurs, for instance, when the epidermis is removed by vesica-



tion, and in cases of herpes zoster. In the early stages of tabes dorsalis the sense of temperature is often abnormally acute. Patients who suffer from other forms of sensory irritation often complain of an intense feeling of burning or of cold, and Brown-Séquard thinks that these sensations are caused by disease of the fibres of the grey substance of the spinal cord, which conduct sensations of temperature. Schiff attributes these sensations to vaso-motor disturbances taking place in parts already rendered hyperæsthetic.

(1) *Causalgia*.—The distressing pain which Weir Mitchell has named *Causalgia* appears to belong to the *thermo-hyperæsthesia*. This pain is variously described by patients as "burning," or as "mustard red hot," or as a "red hot file rasping the skin" (Mitchell). It is generally associated with glossy skin, but often precedes the trophic changes of the skin.

(2) *Dysæsthesia*.—Under the term *dysæsthesia* Charcot describes a sensation of a peculiarly distressing and vibratory character, which ascends towards the central end of the limb, and descends towards its extremity. The sensation is elicited by the slightest touch of the limb or the application to it of a cold body, and it persists several minutes, or even a quarter of an hour after the exciting cause has ceased to act. After some little period of time has elapsed an analogous sensation may be felt at a corresponding point of the limb opposite to the one primarily excited.

(3) *Hyperæsthetic Spots*.—During the paroxysms of lightning pains in locomotor ataxy, circumscribed patches of the skin, generally of the lower extremities, become exquisitely painful to touch. These patches are also subject to attacks of spontaneous pains of a burning character. One patient, in describing this pain, compared it to the sensation that might be caused by rubbing into the skin a burning vesuvian match.

### § 53. *Cutaneous Hyperalgesia.*

Increased sensibility of the common sensations is much more usual than increase of tactile sensibility. In conditions of irritation of the sensory nerves stimuli, which only give rise to the slighter forms of common sensibility in healthy persons, such as tickling, now give rise to pain; and even stimuli, which in



health gives rise to tactile sensations, become painful. The contact of the skin in health with a cold body gives rise to a sensation of temperature, but not to pain; but in conditions of irritation of the sensory nerves such contact gives rise to pain, while the acuteness of the sense of temperature itself is diminished; and the slightest contact with a body, which gives rise to scarcely any sensation of pressure in a healthy person, may become exquisitely painful under these circumstances.

*Lightning Pains.*—Besides the pain caused by the contact of ordinary substances with the surface, intensely distressing pains, occurring more or less spontaneously and in paroxysms, are amongst the most characteristic symptoms of certain spinal affections, as locomotor ataxy. These pains have been described under the names of general neuralgia or neuralgic rheumatism, and are compared by the patients to forked lightning darting through the body; hence they are called lightning-like or lancinating pains. They usually come on in paroxysms, which occur several times in a year or in a month. The paroxysm often begins without any apparent exciting cause, but at other times the attack is induced by bodily exertion, emotional disturbance, the act of coition, gastric disturbances, or variations of temperature.

#### § 54. *Cutaneous Paralgesia.*

(1) *Pruritus.*—Cutaneous pruritus is a sensation which is evidently caused by the action of an abnormal irritation of the nerve ends of the papillæ of the skin, or by a state of undue irritability of these nerve terminations themselves. Pruritus is related to such sensations as tickling, and to burning and stinging pains, but in the former there is an irresistible tendency to scratch the affected part. Pruritus, or itching, is a symptom of various skin affections, and it is a specially prominent feature of scabies and other parasitic affections. Very violent pruritus may also be present in affections of the papillæ in the absence of all cutaneous eruption. Obstinate itching is also a troublesome symptom of diseases in which certain chemical agents are circulating in the blood, as in jaundice and diabetes.

(2) *Formication* is usually caused by an abnormal condition not of the peripheral nerve ends, but of the nerve trunks and central sensory organs. The essential condition which gives

rise to the sensation appears to be some anomaly of the conduction apparatus, which causes the currents to pass through the centripetal fibres, not simultaneously, but in quick succession and with variable intensity. The sensation does not amount to pain, but may be described as feelings of creeping, pricking, or a sensation which has been compared to the crawling of ants. Formication occurs as a transitory symptom in minor mechanical injuries of nerve trunks, such as occurs in the region of distribution of the ulnar nerve to the fingers in contusions of the elbow. It occurs in the foot as a sensation of "pins and needles," or "sleepy sensation," when the sciatic nerve is compressed for some time. Compression of the brachial plexus also causes similar sensations in the upper extremity.

Formication is also met with in diseases of the spinal cord, as tabes dorsalis, and in cerebral affections, as hysteria and hypochondriasis. It may also be produced by poisons circulating in the blood, as morphia, veratrine, ergotine, and probably also the poison of gout.

### § 55. *Neuralgia.*

Neuralgia consists of periodic attacks of severe pain, occurring suddenly and spontaneously in the course of one of the larger nerve trunks, and ramifying in all or a few only of its terminal branches.

(1) *General Symptoms.*—The various forms of neuralgia are preceded generally by premonitory symptoms, which are more or less strongly marked. Slight twitching, formication, pricking sensations, or even pain is felt in the nerve-region about to be affected, and sometimes there is a feeling of general indisposition. The condition of the patient at the time of the first attack is always, as Dr. Anstie has pointed out, one of *debility*, either general or special. Patients are frequently attacked for the first time after an exhausting illness or fatigue, or when they are in an anxious condition from some cause or another.

There is always a degree of suddenness in the onset of neuralgia. Usually the first warning is a sudden, not very severe, and transient dart of pain. The patient has probably been suffering from some degree of general fatigue and malaise, and the skin of the affected part has been somewhat numb,



when a sudden stitch of pain darts into the nerve. It ceases immediately, but in a few seconds or minutes returns, and then darts of pain recur more and more frequently, until at last they blend together, so that the patient suffers continuous and violent pain for a minute or so, then experiences a short intermission; but the pain returns again, and so on. These intermittent spasms of pain go on recurring for one or several hours, then the intermissions become longer, the pains abate, and at last the attack wears itself out. The intensity of the pain during the paroxysm may vary from moment to moment, and it may become so intense that the patient is almost driven to desperation.

The pain is described as tingling, tearing, boring, stabbing, dragging, burning, or lightning-like, and it may be felt as if it were on the surface or deep in the bones. In some cases the pain shoots from the centre to the periphery, forming descending neuralgia; while at other times it takes the reverse course, forming ascending neuralgia. The locality of the pain varies: sometimes it is in a fixed spot, while at other times it changes about; and it is often directly referred to a nerve-trunk which is painful throughout its whole course. The pain of neuralgia is usually increased by movement, so that the patient keeps the affected part as much as possible at rest, although occasionally it is relieved by movement of the part. Superficial irritation of the skin frequently produces an attack of pain, but continuous firm pressure on the part relieves the pain.

(2) *Painful Points*.—These points were first described by Valleix, under the name of "*points douloureux*." An examination of the part during an attack of superficial neuralgia will reveal one or more points which are extremely sensitive to the pressure of the tip of the finger. The sensitiveness of these points stands almost in a direct relation with the severity of the paroxysm, but occasionally they may be present during the period of remission; in some instances pressure on them produces an attack. These tender spots are found at various points in the course of the affected nerves, where their trunks pass from a deeper to a more superficial level, and especially where they emerge from body canals or pierce fibrous fasciæ, or even when the nerves lie on a hard bed, so that they may be easily compressed.



(3) *Points Apophysaire*.—Trousseau insists that in all forms of neuralgia the spinous processes of the vertebrae corresponding to the origin of the painful nerve, and which he calls "*points apophysaire*," are painful on pressure; but these points are also present in cases of "spinal irritation" and in myalgia. Anstie found that the painful points were absent in the early stage of neuralgia, and only made their appearance at a subsequent stage and at the situation which had been the focus of the neuralgic pain.

(4) *Concomitant Sensory Symptoms*.—During the height of the neuralgic paroxysm there is frequently an *irradiation of the pain* to other sensory nerves, generally to branches of the same trunk or to neighbouring nerves, but occasionally to more or less distant nerves. When, for instance, one branch of the fifth is primarily affected, the pain spreads to the two others; next in frequency from one nerve as the sciatic to the corresponding one on the opposite side; and lastly to quite different nerve territories, as from one of the intercostal nerves to the fifth. These radiated pains are not usually so intense as the original one; the duration of the attack is not so long; they begin to be felt when the paroxysm has reached its height, and disappear before it has completely subsided. Various subjective sensations, as formication, creeping, and numbness, are felt in the region to which the affected nerve is distributed during the remission; but during the paroxysm their presence is obscured by the severity of the pain. More marked disturbances of sensibility, as hyperæsthesia or anæsthesia, are also frequently met with in the affected region (Türk, Trousseau, Traube, and Anstie). Nothnagel has recently found that in neuralgia of the nerves of the extremities, without any demonstrable anatomical lesions, an alteration of the tactile sensibility of the skin is invariably present. As a rule, in recent neuralgia having a duration of from two to eight weeks, there is *hyperalgesia* of the skin, and in neuralgia of long standing *anæsthesia*. These symptoms are usually limited to the region of distribution of the affected nerve; but in some cases of limited neuralgia the disturbances of sensibility affect the whole of that side of the body.

(5) Various *motor, vaso-motor, secretory, trophic, and psychical*

symptoms are associated with neuralgia, but our main object at present is to describe the elementary symptoms separately; hence the description of the remaining concomitant symptoms of neuralgia may be deferred at present.

(II.)—SENSORY AFFECTIONS OF THE VOLUNTARY MUSCLES  
(MUSCULAR ÆSTHESIONEUROSES).

§ 56. *Muscular Sensibility and Muscular Sense.*—There can be no doubt that certain sensations arise during muscular activity, which appear to be quite independent of the cutaneous sensibility. These sensations are of two kinds: the first, called *muscular sensibility*, corresponds with the common cutaneous sensation, and consists of the pleasurable and painful feelings connected with muscular exercise; and the second, called *muscular sense*, corresponds with the cutaneous tactile sensibility, and consists of those sensations by which the mind is enabled to discriminate differences in the degree of contraction of the muscles; the former being an emotional and the latter an intellectual sensation.

(1) *Tests for Muscular Sensibility.*—The state of muscular sensibility is best tested by means of faradisation. When a healthy muscle is made to act a dull feeling accompanies the contraction, which is entirely absent if the muscular sensibility is abolished. There is also absence of sensitiveness when heavy pressure is made upon the muscles. This form of muscular anæsthesia may occur with or without cutaneous anæsthesia, but it is usually accompanied by muscular paralysis. The sense of muscular effort may remain intact.

(2) *Tests for Muscular Sense.*—To test the muscular sense the patient should be made to lift various weights and to form an estimate of their differences. In order to eliminate the cutaneous sense of pressure the weights should be placed in a cloth and suspended from the limb to be investigated, or it may be held in the fingers, so that it is supported rather by friction than by pressure. The sense of effort may also be tested by the dynamometer. The patient may also be made to move the limb into certain prescribed positions with closed eyes, or he may be asked, the eyes being still kept closed, to touch a particular part of the body, such as the tip of the nose or the lobule of an ear, with the forefinger of one hand, or to take hold of a ticking watch held before him; or, in the case of the legs, to describe an imaginary circle on the floor with the big toe; and all these actions will be either not accomplished or imperfectly performed, according as the muscular sense is abolished or to the degree of its impairment.



§ 57. *Muscular Hyperæsthesia and Hyperalgesia* are often met with in diseased conditions. The excessive feeling of fatigue and prostration, which occurs on slight exertion in the prodromal stage of acute diseases, is probably due to excessive irritability of the nerves of common muscular sensibility; hence this condition may be regarded as one of muscular hyperalgesia. The feeling of unrest and desire of constant change of position, which has been called the fidgets, and which is so troublesome to nervous and hysterical patients, is due to muscular hyperæsthesia.

It is not improbable that muscular hyperæsthesia may play a part in the production of certain spasmodic diseases, such as writers' cramp, and in choreic conditions. In spasmodic wry neck and other spasmodic affections, as well as in muscular cramps, the hyperæsthetic condition of the affected muscles is often very great, giving rise to intense pain. Painful conditions of the muscles occur, which have been called myalgia or myodynia. It is very probable that myalgia is sometimes due to morbid irritative changes in the muscle itself; but the fact that the pain occurs in individual muscles, and with paroxysmal exacerbations side by side with cutaneous neuralgia, such as sciatica, or along with general hysterical symptoms, shows that the affection is at times of nervous origin. This condition is most frequently met with in the muscles of the neck and of the lumbar region; and inasmuch as it closely corresponds to cutaneous neuralgia, and to the neuralgia of joints or arthralgia, it might well be called *muscular neuralgia* or *myoneuralgia*.

§ 58. *Muscular Anæsthesia* is by no means an uncommon affection. The common sensibility, as tested by the faradic current, or the electro-muscular sensibility, may be diminished or abolished, while the electro-muscular contractility and the electro-cutaneous sensibility remain intact.

Duchenne drew attention to the fact that electro-muscular contractility may be unaffected; while both electro-muscular sensibility and voluntary power are abolished. This occurs more especially in hysterical paralysis. In other cases of hysteria voluntary power is maintained, while electro-muscular sensibility is abolished. Loss of muscular sensibility may occur while the muscular sense is retained, and the former condition may be conveniently termed *muscular analgia*.

*Anæsthesia* of the muscular sense, or of the sense of muscular effort, is characterised by the diminution or loss of the capacity of recognising small weights, or of perceiving differences of weight by muscular contraction. If the muscles are



completely paralysed, no test of the muscular sense can be applied. The loss of the muscular sense is frequently associated with absence of the feeling of equilibrium of the body, inability to determine when the eyes are closed the position of the limbs, or the extent of any movement that may be performed. It is, however, not easy to determine how far these functional disturbances are dependent on the mere loss of muscular sense; since other sensory impressions, such as those of the skin, bones, and joints, participate in these actions.

(III.)—SENSORY AFFECTIONS OF THE JOINTS AND BONES  
(ARTICULAR AND OSSEOUS AESTHESIONEUROSES).

The existence of sensory nerves in the bones and joints has not yet been determined anatomically, but the facts of pathology leave no room for doubt that such nerves must exist. The sensibility of the bones and joints is apparently only very slight in the normal condition, but they may become exquisitely sensitive under pathological conditions. It is probable that the normal sensibility of the bones and joints play an important part in enabling us to determine with closed eyes position in space and the position of our limbs after passive movements.

§ 59. *Osteoneuralgia*—*Arthroneuralgia*.—Excessive or abnormal sensations occur in the bones and joints, usually in the form of pain, and when the pain is not caused by recognisable anatomical changes in those parts, the condition is regarded as neuralgia of the bones and joints, which may respectively be called *osteoneuralgia* and *arthroneuralgia*.

Neuralgia of the bones and joints differs from cutaneous neuralgia in not radiating along the course of the principal branches of the affected nerves. Neuralgia in the region of distribution of the nerve-trunk, such as the sciatic, is not usually associated with neuralgia of the bones and joints; while conversely, the latter kind of neuralgia is not usually associated with neuralgia in the region of distribution of the neighbouring cutaneous nerves. It is evident, therefore, that sensory nerves of the joints and bones do not belong to the system of afferent fibres of the mixed nerves, and it is probable that they reach the bones along with the sympathetic plexus which surrounds the vessels. The fact that neuralgia of the joints is frequently associated with local disturbances of the circulation, such as redness, heat, and œdema, also appears to favour this supposition. But certain cutaneous neuralgias, such as the lightning and

burning pains of locomotor ataxia, are often associated with neuralgic pains in the bones and joints, but in all such cases the irritation which is the source of the neuralgic pains is situated in the posterior root of the nerves, and in their continuation through the posterior columns to reach the posterior horns of grey matter.

§ 60. *General Symptoms of Arthron neuralgia.*—The essential symptom of articular neuralgia is intense pain. The pain occurs in the joint and surrounding dense structures in paroxysms which come on spontaneously, and which are separated by intervals of complete or comparative freedom from pain. The quality of the pain offers little which is characteristic. At times it is described as tearing or shooting through the joint like lightning; at other times as boring or stabbing pain. Various other abnormal sensations may be felt besides the pain, such as a sensation of heat or of cold, numbness, and formication. The attack of arthralgia is frequently ushered in by premonitory symptoms, consisting of abnormal sensations in the skin. Pressure, as a rule, increases the pain; but, as occurs in cutaneous neuralgia, slight and superficial pressure may produce intense pain; while deep, continuous, and uniform compression produces no effect, or even relieves the pain. The pain is also much increased when the attention of the patient is directed to it; and on the other hand it is diminished under the influence of general fatigue, and does not prevent the patient from sleeping. Painful points may be obtained about the affected joint on pressure, which Esmarch and Berger have endeavoured to determine for individual joints. These probably correspond to the points where small branches of sensory nerves pass to the capsule of the joint along with the vessels. Cutaneous hyperæsthesia is very generally present in the early stages of the affection in the neighbourhood of the affected joint; while a diminution of the sensibility of the corresponding portion of the skin may be present in cases of long standing. Besides the local periarticular pressure points, other tender spots not unfrequently occur in the course of the affection as over neighbouring nerve trunks, or over the spines of the corresponding vertebræ.

In many cases the sensory disturbances are associated with motor, vaso-motor, and trophic affections. The motor distur-



bances consist of weakness and helplessness in the use of the affected joint, owing to the fear of inducing a paroxysm of pain; but occasionally spastic contraction of the muscles surrounding the joint occurs, which is increased by all attempts to move the joint, but completely disappears under chloroform. Sometimes a creaking or crepitating noise is heard when the joint is moved, the cause of which is not fully ascertained. When a definite group of the muscles round the joint, as the extensors, become contracted, their antagonists may undergo a certain amount of atrophy, but seldom to any great extent.

The vaso-motor disturbances, consisting of redness, heat, and increased secretion of sweat, sometimes occur in the neighbourhood of the affected joint. These phenomena may appear and disappear very rapidly; and may, like the paroxysms of pain, assume an intermittent character, at times of regular type, the symptoms, for instance, recurring every evening. A circumscribed doughy or fluctuating swelling in the neighbourhood of the affected joint, associated with redness and increased temperature, is sometimes observed, which was compared by Brodie to an unusually large urticaria wheal.

Swelling of the joint from serous effusion within the capsule may take place, and it is apt to be regarded as of inflammatory origin, while effusion of the tissues surrounding the joint as the result of irritating applications tends still further to obscure the true nature of the affection.

#### (IV.)—SENSORY AFFECTIONS OF THE INTERNAL ORGANS (VISCERAL ÆSTHESIONEUROSES).

Visceral neuralgia has already been considered generally, along with cutaneous neuralgia, but various other forms of visceral sensory disturbances remain to be considered. These forms are visceral hyperæsthesia, paralgesia, and anæsthesia.

§ 61. *Visceral Hyperalgesia and Paralgesia.*—The visceral hyperalgesia and paralgesia depend partly upon abnormal irritation of the visceral sensory nerves, or upon increased irritability of these nerves. These sensations belong to the sphere of common sensations, since they have no distinct objective



character; in other words, they are related to the feelings and not to the intellect. The most usual feelings which come under this category are titillation, globus, pyrosis, bulimia, polydipsia, abnormal feeling of voluptuousness, and the feeling of oppression.

(1) *Titillation* is a sensation which is induced by abnormal irritation or undue irritability of the nerve ends of certain regions of the respiratory mucous membrane, and corresponds with pruritus of the external skin. And as the latter leads to an irresistible tendency to scratching, so the former leads to the reflex respiratory movements which produce coughing. The sensory branches of the vagus, and especially the superior laryngeal branch, are those which are irritated during coughing.

(2) *Globus* is a sensation in which the patient complains that a ball ascends from the epigastric region to the throat. This sensation is either one of the symptoms of hysteria, or it may constitute an epileptic aura. This feeling is supposed by some to depend upon spasm of the œsophagus and pharynx, and it has consequently been called *Œsophagismus*. This explanation is, however, very unsatisfactory.

(3) *Pyrosis*, or *waterbrash*, is a painful sensation in the epigastrium, consisting of a sense of burning, generally attended with the rising of a quantity of clear watery fluid into the mouth, which may be tasteless and neutral, or sour and acid in reaction. An attack of pyrosis may last from a few minutes to many hours, with alternating remissions and exacerbations.

(4) *Bulimia* is a feeling of hunger, which is abnormal in its period of occurrence or in its intensity, and which is appeased only for a short time by taking food. In many cases a large quantity of food must be taken before the feeling of repletion is reached; but in order to constitute bulimia it is necessary that the sensation of hunger should return in an unusually short time after it has been satisfied. At times a small quantity of food satisfies the feeling of hunger; but if the latter returns after an interval of one or two hours, this constitutes bulimia. It must be assumed that in bulimia the afferent nerves, irritation of which constitutes the feeling of hunger, are in a condition of abnormal irritability. It may be

said that the absolute hunger minimum is in such cases greatly diminished, just as the absolute pain minimum is in cutaneous neuralgia. Bulimia is frequently of central origin, and often occurs in hysterical and neuropathic subjects. It also occurs in epileptoid conditions, in diabetes, and during convalescence from exhausting diseases, and is a frequent symptom of insanity.

(5) *Polydipsia* is an excessive feeling of thirst, and, like bulimia, it must be regarded as a hyperalgesia. It is generally believed to be due to increased irritability of the sensory branches of the vagus; but this is very doubtful, since the sensory branches of the mucous membrane of the mouth and throat, including branches of the fifth and glosso-pharyngeal nerves, participate in the sensation of thirst. Polydipsia is a constant symptom of polyuria and diabetes; and, like bulimia, it comes on after exhausting diseases, and as a symptom of hysteria.

(6) *Excessive Voluptuous Feeling* was ascribed by Romberg and others to neuralgia of the spermatic plexus. Voluptuous sensations are specially frequent in the female sex, but they are by no means relatively more frequent in the hysterical. In the narrated cases in which excessive voluptuous feelings were observed in hysterical subjects these were associated with excessive reflex and psychical phenomena, with quickening of the respiration and pulse, great emotional disturbance, and even loss of consciousness and general convulsions. In women who are not hysterical excessive voluptuous feelings are found associated with symptoms of nymphomania, or they appear as precursors of an epileptic attack. In a large number of cases local irritation of the external genitals is the cause; while in other cases the abnormal sensations appear to depend upon changes in the afferent conducting apparatus of the genital organs in its course through the spinal cord or brain. It is very difficult to classify the cases which have been adduced as examples of increased voluptuous feelings in men. In many cases this symptom is associated with frequent but incomplete erections, premature ejaculation of semen, and more or less complete impotency. Local irritation of the genital apparatus is usually the cause of these abnormal sensations, but at times the cause may be spinal, as in *tabes dorsalis*.



(7) *Feeling of Oppression*, when general, arises probably from over work or deficient nourishment of the nervous system. It is described by the patient as a feeling of heaviness, dulness, and depression of spirits. A more specific form of oppression occurs in connection with cardiac affections, as angina pectoris. A feeling of oppression or constriction is experienced across the chest as if it were being forcibly compressed, and it is attended by a sense of suffocation. The patient also suffers from an intense feeling of impending danger or threatened death. Peripheral irritation of either the cardiac, solar, or mesenteric plexus may probably give rise to this feeling at times, although it is more commonly met with in connection with affections in the region of distribution of the cardiac plexus.

§ 62. *Visceral Analgesia*.—Very little is known with respect to visceral analgesia. The normal functions of the viscera are performed without much sensibility; although visceral sensations contribute greatly to the feeling of well-being and comfort. It is probable, therefore, that diminution of visceral sensations contribute in an equal degree to our general feeling of bodily discomfort. It is difficult to apply any reliable tests to determine the degree of visceral sensibility. The degree of reflex irritability of the sympathetic has been proposed as a test of the degree of its sensory irritability; but this test is liable to two manifest objections. In the first place, it is questionable whether the peristaltic action of the bowels and similar movements are really of a reflex nature, and are not due to irritation of automatic peripheral ganglion-cells. In the second place, even if it be proved that the movements presided over by the sympathetic are of a reflex nature, it by no means follows that because the reflex conduction is diminished or abolished that the conduction through the afferent sensory fibres is also affected. The reflex tonus of the constrictor muscle of the urethra may be abolished, giving rise to incontinence of urine in the absence of any affection of the sensibility of the bladder, although it is by means of the afferent nerves of the bladder that the reflex tonus is maintained. On the other hand, anæsthesia of the bladder may occur without the reflex tonus of the sphincter of the urethra being diminished, and it

may even be increased under such circumstances. Similar relations obtain in other reflex phenomena, as in erection, and the secretion and ejaculation of semen.

(1) *Anæsthesia of Laryngeal and Bronchial Branches of the Vagus*.—Anæsthesia occurs in the territory of the laryngeal and bronchial branches of the vagus when the normal amount of irritation does not give rise to the feeling of titillation and to the reflex movements of coughing. This condition may assume a very grave significance when catarrhal secretions cease to induce cough, and consequently accumulate so as to cause suffocation. In such cases the anæsthesia appears to be of central origin, and it is often both a result and a cause of increasing poisoning by carbonic acid. In some cases the inspiration is abnormally slow without any subjective feeling of inspiratory desire being excited. Such cases are probably due to a certain degree of anæsthesia in the region of distribution of the vagus.

(2) *Anæsthesia in the territory of the gastric branches of the Vagus* gives rise to *polyphagia*, a condition in which an unusual quantity of food must be taken before the feeling of hunger is satisfied; or in which the feeling of repletion is never obtained, however much food is taken. The experiments of Legallois, Brachet, and others have proved that, on section of the vagi, animals continue to eat until the œsophagus is filled with food. Pathological observations also confirm this conclusion. Swan mentions a case where the patient could not experience the feeling of repletion after eating large quantities of food, and where after death both vagi were found atrophied and disorganised. This condition often appears in affections of central origin, as hysteria, epilepsy, and various forms of insanity.

(3) *Anæsthesia of the Sexual feelings* is most frequently observed in the female sex. Diminution of these feelings is more frequent in hysterical females than excess of voluptuous sensations; and this is not unfrequently associated with decided aversion to coitus. Complete absence of voluptuous feelings is probably due to anæsthesia of the mucous membrane of the genitals; such as occurs in hysterical females in association with diffuse or circumscribed cutaneous anæsthesia. Analogous con-



ditions are observed in the male sex, as a result of sexual excesses and onanism, or as a symptom of chronic affections of the spinal cord, such as spinal meningitis and tabes dorsalis, or at times in the absence of any appreciable cause. In these cases diminution of the electric sensibility of the glans penis and of the external genitals may be detected. The power of erection and the secretion of semen become diminished or abolished in consequence of the diminution of the reflex irritability, and these conditions have respectively been designated *Impotency* and *Aspermatism*.

(V.)—SENSORY DISTURBANCES OF THE SPECIAL SENSES.

The consideration of this subdivision of the *Æsthesioneuroses* is reserved for the special part of the work.

## CHAPTER V.

## II.—ELEMENTARY AFFECTIONS OF INDIVIDUAL MOTOR MECHANISMS (KINESIONEUROSES).

## (I.)—MOTOR DISTURBANCES OF THE STRIPED MUSCLES (EXTERNAL KINESIONEUROSES).

THE striped muscles may be excited to contraction either by voluntary, reflex, or automatic excitation. Any deviations from the normal motor reactions obtained by the application of various stimuli constitute disease; but these abnormal deviations can, as a rule, only be relied upon as evidence of disease when they are elicited in response to a methodical examination. Our first object must therefore be to give a succinct account of the various methods adopted for the examination of the phenomena presented in disease of the nervous mechanisms which co-ordinate the movements of the muscles of external relation.

§ 63. *Methods of Examining the Motor Apparatus.*

1. *General Examination.*—Motor disturbances are recognisable partly in consequence of certain positions and movements being too strongly marked, as in cramps, and partly by feebleness of execution or inability to perform certain movements, as in paralysis, and the consequent deformities produced in the general symmetry of the body. An exact knowledge of the anatomical connections and functions of the different muscles is necessary for the recognition of those which are affected, and this is more especially necessary in spasmodic affections. In spasm of deeply-seated muscles which act in association with others, it is very difficult to discriminate the muscles affected. A complete and minute examination of the whole naked body, the performance of active and passive antagonistic movements, and the forcible extension of the abnormally placed parts of the body, are the principal means of investigation. Valuable information is afforded by local faradisation of the muscles which can be reached. An



artificial spasm can by this means be produced in the corresponding muscles of a healthy individual, and the deformity produced by the artificial spasm can be compared with that produced by the morbid spasm. Accurate conclusions may be drawn from the faradisation of antagonistic muscles and of the symmetrical muscles of the other half of the body.

2. *Special Examination.*—The patient must also be directed to perform voluntary movements, such as standing, walking, or writing, as well as the mimetic movements that occur in speaking and laughing, so that any deviation from the normal movements may be carefully observed.

When the paralysis is incomplete it becomes important to ascertain the degree of motor impairment. The force capable of being exerted by certain groups of muscles can be measured directly by means of the various forms of *dynamometer*; and an approximate estimate may be made by comparative testing of the resistance which can be opposed to passive movements. It is also important to observe whether the muscular contraction can be maintained for some time without inducing exhaustion.

It must also be observed whether the contractions of the different muscles are harmoniously combined, so as to produce the various movements with accuracy and precision, or whether they are accompanied by trembling or disturbing secondary movements. With this view the patient must be asked to perform special movements, such as those concerned in drawing straight lines or circles, touching particular points with the fingers. Valuable information is also afforded from the observation of various complicated movements, such as those of running, hopping, ascending a stair, writing, sewing, and knitting.

The performance of passive movements enables us to judge whether the muscles are completely relaxed, tense, or contracted; or whether they are readily thrown into a state of convulsive tremor.

3. *Cutaneous and Tendinous Reflex Stimuli.*—The condition of the reflex movements of the skin, accessible mucous membranes, and organs of special sense, must be tested by tickling, pricking, pinching, faradic excitation, and by the special excitants of the special senses.

Reflex actions are also induced under certain conditions by tapping tendons, the periosteum, fasciæ, and probably the joints. The extensors of the leg on the thigh may be thrown into contraction by a smart tap on the tendon of the patella; and this state is enormously increased in some diseases, while in others it is abolished.

4. *Mechanical Contractility.*—Mechanical stimuli are also useful in testing the condition of the muscles. When a muscle is removed from the body the irritability gradually diminishes, and after a time disappears altogether. But if a sharp blow be struck across a muscle which has entered into the later stages of exhaustion, a wheel lasting for several seconds is developed. From this wheel small waves of contraction run in both directions towards the extremities of the muscles, and this form of muscular action has been called "idio-muscular" contraction, because it may be brought out when ordinary stimuli have ceased to produce any

effect. A moderately strong blow over almost any muscle of the body, under normal conditions, induces a contraction of the fasciculus struck, especially if the blow fall near the point of entrance of the motor nerves. These contractions occur with greater readiness in the pectoralis major, deltoid, and the extensor muscles of the forearm, than in the other muscles of the body; and in exhausting diseases, such as phthisis, the slightest tap over the pectoral muscles causes a circumscribed tumour due to local contraction of the subjacent muscle.

#### AKINESIS OF THE MUSCLES OF EXTERNAL RELATION

§ 64. The most prominent symptom of motor paralysis is the inability to contract certain muscles by voluntary effort. The paralysed limbs hang motionless, or they are weak and helpless if the paralysis is incomplete. The utmost variety may occur in the distribution and extent of the paralysis. It is sometimes limited to a single muscle, or group of muscles; at other times all the muscles supplied by a particular nerve or plexus of nerves may be implicated; while in other instances all the muscles of one extremity may be paralysed. In other cases the paralysis affects both halves of the body symmetrically, and then it generally begins in the lower extremities and spreads to the trunk and upper extremities. This is the usual kind of paralysis from disease of the spinal cord, and is termed *paraplegia*. In other cases the paralysis affects the lateral half of the body, implicating the face, arm, and leg of the same side, and it is thus termed *hemiplegia*. The lesion which causes this form is usually situated in the opposite hemisphere of the brain, although hemiplegia of spinal origin is not unknown.

The condition of the paralysed muscles varies in different instances. The muscles are sometimes completely flaccid and relaxed, and destitute of the faintest trace of tone or contractility; in other cases they are more or less tense and contracted; while in still others they are at first relaxed and become subsequently contracted. If the condition be one of incomplete paralysis or paresis, the movements which can still be performed are not unfrequently accompanied by trembling and uncertainty of execution. Loss of voluntary power does not necessarily entail loss of reflex contractility. In cerebral paralysis, and in paralysis due to disease of the antero-lateral columns of the cord, the reflex contractility is wholly unaffected, and in some cases it is



even increased. Complete absence of all reflex movements only occurs when there is interruption in the conduction in the peripheric motor nerves, or in the spinal cord itself, or when the muscles are destroyed. The automatic movements are also unaffected in cerebral paralysis. The automatic movements of respiration are more liable to be affected in spinal disease. The automatic movements of the iris may be implicated either in disease of the upper portion of the spinal cord—the region termed the cilio-spinal centre—or where a cerebral lesion injures the nucleus of origin of the third pair of nerves. Paralysis from lesion of the cilio-spinal centre induces contraction of the pupil, or *myosis*, and that from lesion of the third nerve, dilatation or *mydriasis*.

The functions of the rectum and bladder may be interfered with in various forms of paralysis, but they are more specially liable to be implicated in diseases of the spinal cord.

§ 65. *Associated Symptoms*.—When the paralysis is of centric origin certain *associated* movements may be preserved in the paralysed parts, but these will be described under the name of *synkinesis*.

*Sensory disturbances* are very frequent concomitants of every form of paralysis. When a mixed nerve is affected the sensory paralysis is generally equal in extent to the motor paralysis. The motor paralysis is, however, usually more marked in degree than the sensory paralysis, and recovery of sensation is generally more speedy and certain than of motor power. Violent neuralgic pains, hyperæsthesiæ, and paræsthesiæ are frequently associated with all forms of motor paralysis, except that which arises from lesion of a purely motor nerve.

The *vaso-motor* and *trophic* symptoms, which are so often associated with paralysis, will be subsequently described.

§ 66. *Electrical Stimuli* are also very valuable for testing the reflex irritability, but they are still more important, if possible, when directly applied to the investigation of the motor apparatus. Two kinds of electric currents are usually employed for the purposes of electrical research—the faradic, induced, or interrupted current, and the galvanic, constant, or continuous



current. Faradic currents consist of a series of isolated currents, each of momentary duration, and of very rapid development and decline, following each other in quick succession and flowing alternately in opposite directions. Galvanic currents run in the same direction and with the same intensity, and are continuously produced. By means of the commutator, however, the current can be interrupted at pleasure, and thus closing and opening muscular contractions may be induced. The current may also be rapidly reversed by means of the commutator, a method which induces a very powerful contraction.

1. *Faradic Excitability or Irritability* is the term used to designate the kind and strength of the reactions exhibited by muscles under the influence of the faradic current. Muscular contraction may be induced by the *direct* application of the faradic currents to the muscles themselves, or *indirectly* through excitation of the motor nerves. For the purpose of testing the faradic excitability the current obtained from the secondary coil of the ordinary induction apparatus called the "secondary induced current" is generally used, although the current from the primary coil, called the "primary induced, or extra current," may also be employed.

The cathode of the secondary induced current is usually employed as the exciting pole, while the anode may be placed upon some indifferent part of the body, as the sternum, or patella. Excitation of the muscles through the accessible motor nerves with a feeble faradic current induces a minimum contraction; whilst on the strength of the current being increased, strong tetanic contractions ensue. In comparing the results obtained in different parts of the body, it is of importance to remember that in health the muscles supplied by the symmetrical nerves of the two sides of the body can be excited to minimum contraction by the same strength of current, and that those supplied by various superficial nerves, such as the frontal branch of the facial, the spinal accessory, ulnar, and peroneal nerves, can also be excited to minimum contraction by currents of the same intensity. Any considerable deviation from these conditions must be regarded as pathological.

Direct excitation of accessible muscles is best performed when the poles are applied over the points at which the motor nerves enter the muscles, and these may be ascertained by reference to the diagrams reproduced from Ziemssen, in the special part of this work.

2. *Galvanic Excitability* is a term used to express the reactions obtained in response to opening and closing the circuit and to the continuous passage of the galvanic current. Either the anode or the cathode may be used as the exciting pole, whilst the other is applied to some indifferent part of the body, as the sternum.

The law of contraction of both motor nerves and muscles rests upon the facts that the cathode produces contraction chiefly on closure of the

current; the anode chiefly on opening the current; and that the stimulus of the cathode is stronger than that of the anode. The reactions obtained with different strengths of current may be deduced from these fundamental facts.

With a weak current the cathode produces simple contraction on closure of the current, while there is no reaction from the anode; with a current of medium strength the cathode produces stronger contraction on closure of the current, but no opening contraction; while the anode causes feeble contractions both when the current is closed and when it is opened.

With a strong current the cathode produces on closure of the current a tetanic tonic contraction, and a feeble contraction on opening the current; while the anode produces lively contraction both on opening and closing the current.

The law of contraction may be expressed by the following formulæ:—  
Let An=anode, Ca=cathode, C=contraction, c=feeble contraction,  
C'=strong contraction, S=closure of current, O=opening of current,  
Te=tetanic contraction; then—

Weak currents produce Ca S C.

Medium " " Ca S C', An S c, An O c.

Strong " " Ca S Te, An S C, An O C, Ca O c.

In diseased conditions deviations from the normal law of contraction may occur by way of excess or diminution of the excitability; or by changes in the quality of the various reactions. These quantitative and qualitative changes will be subsequently described.

§ 67. *Electrical Examination* of the nerves and muscles is necessary in making a precise diagnosis of the several forms of paralysis, and in forecasting the progress of the case either towards recovery or towards an irremediable condition. The results obtained may be subdivided into several groups:—

1. *The First Group* comprises those cases where both nerves and muscles react normally to faradic and galvanic currents. The electrical contractility is as a rule unaffected in paralysis of cerebral origin and in many forms of spinal paralysis, as in chronic myelitis, and even in several kinds of peripheral paralysis, as in slight traumatic paralysis and in that of rheumatic origin.

2. *The Second Group* includes the cases in which there are simple quantitative changes of the electrical excitability, manifested either by increase or diminution of the normal reaction.

(a) Simple increase of the electrical excitability may be manifested in several ways. If the faradic current be applied to the nerves and muscles there is an increase of the amount of contraction with the same strength of current, or there is an increase of the distance of the secondary coil



at which minimum contractions are produced. If the galvanic current be employed, a cathodal closing powerful contraction (Ca S C') occurs with feeble currents; and a cathodal closing tetanus (Ca S Te) is induced when the strength of the current is slightly augmented; and an anodal opening contraction (An O C) is caused by weak currents. A cathodal opening contraction (Ca O C) is also readily established, and in some cases an anodal opening tetanus (An O Te) may be induced.

Simple increase of electrical excitability occurs to a moderate extent in certain forms of cerebral and of spinal paralyses; and in some cases of tabes dorsalis; and even in a few cases of peripheral paralysis it occurs as a transient symptom.

(b) Simple diminution of electric excitability is mainly manifested by the reactions to the various currents being the reverse of what they were in simple increase of the excitability. When the faradic current is applied to the nerves or muscles the same strength of stimulus causes a weaker contraction than in health, or there is a diminution of the distance of the secondary coil at which minimum contractions are produced, which may in some cases proceed to complete extinction of faradic contractility. With galvanic currents cathodal closing tetanus (Ca S Te) first disappears and cannot be induced by any ordinary strength of current; anodal closing and opening contraction (An S C and An O C) then disappear, and by and by cathodal closing contraction (Ca S C) can only be obtained with the strongest currents, and ultimately there is complete loss of galvanic excitability.

Simple diminution of the galvanic excitability is rare in cerebral paralysis, but occurs in the later stages of bulbar paralysis and in certain forms of spinal and peripheral paralyses. It may indeed be laid down as a general rule that whenever the nervous lesion is such as to give rise to simple atrophy of the paralysed muscles, there is simple diminution or complete extinction of the electric excitability, unaccompanied by any qualitative changes in the reactions obtained.

3. *The Third Group* consists of what Erb has proposed to call the "reaction of degeneration," and includes both *qualitative* and *quantitative* alterations of electrical excitability. The alterations in the reactions of the nerves and muscles do not run a parallel course, so that the two must be separately described.

(a) *Reaction of the Nerves*.—The alteration in the reaction of the nerve begins on the second or third day after the attack of paralysis. A continuous uniform diminution of both the faradic and galvanic excitability is observable without any qualitative change, and in very rare cases only is it preceded by slight increase. The diminution begins in the part nearest the lesion, and extends rapidly to the periphery. At the end of the first, or in the course of the second week (from the seventh to the twelfth day), the excitability wholly disappears. In incurable cases the loss of the excitability is permanent; but if repair of the diseased tissue takes place, the excitability, after being lost for a variable period, is

gradually restored. The reactions to both currents reappear almost simultaneously, beginning first in the central segments of the nerve and spreading slowly to the periphery. The reactions at first obtained are very feeble, but they gradually increase in strength as repair proceeds, although remaining a long time below the normal standard even after the restoration of voluntary power appears complete. During the early stages of regeneration voluntary movements may be effected through the paralysed nerves at a time when they give no reaction to the electrical stimulus, the duration of this period being from a few days to several weeks. Erb has found in experiments on animals, and in traumatic paralysis in man, that in those cases where voluntary impulses are conducted through the injured nerves the electric stimulus will also induce contraction if it be applied above instead of below the point of injury.

(b) *Reaction of Affected Muscles.*—The electric reactions of the paralysed muscles are much more complicated than those of the degenerated nerves, since they are not affected in the same manner by the two currents. The reactions obtained by the faradic are simpler than those obtained by the galvanic current, since the former are almost entirely similar to those obtained by the application of the current to the degenerated nerves. When the paralysed muscles are exposed to the faradic current a diminution of the excitability is observed towards the end of the first week, and usually there is complete extinction towards the end of the second week. When the electrodes are applied to the surface of the body, although feeble contractions may be obtained by electrical acupuncture, they are limited to the fasciculi directly excited. When the case is incurable the faradic contractility becomes permanently abolished; but when there is a return of voluntary power the faradic contractility of the muscle reappears, although usually somewhat later than in the nerves. As recovery proceeds the faradic excitability increases gradually and slowly, and generally remains for a long time abnormally low, especially if the paralysis has been of long duration.

*The Galvanic Excitability* falls in conformity with the faradic excitability during the first week, but in the course of the second week the former is remarkably increased, and continues to increase during the following few weeks. The affected muscles now react to currents so feeble in intensity as to be entirely inoperative on healthy muscle, and there is a remarkable change in the quality of the contraction induced. The contractions induced by the galvanic current in healthy muscle are short and lightning-like; but in the paralysed muscles they are slow and protracted, and even those induced by feeble currents readily pass into a muscular tonus, which continues the whole time the current is transmitted.

*The Law of Muscular Contraction* also becomes changed along with the increase of the excitability. There is a gradual and strong increase of the anodal closing contraction, so that it soon equals or exceeds the cathodal closing contraction ( $An\ S\ C = or > Ca\ S\ C$ ). The cathodal opening contraction on the other hand increases in a relatively greater degree than



the anodal opening contraction ; so that the former soon equals or exceeds the latter ( $\text{Ca O C} = \text{or} > \text{An O C}$ ) ; hence here is complete inversion of the normal formula of muscular contraction. When the alteration has attained a high degree the opening contractions disappear, and this condition lasts, though with some variations in different cases, for from three to eight weeks, or sometimes for a much longer period.

The increased galvanic excitability now diminishes, and stronger and stronger currents are required to produce contraction ; and in incurable cases an extremely feeble anodal closing contraction is usually the last sign of the disappearing muscular irritability. When, on the other hand, regeneration and recovery take place, the normal mode of reaction is gradually re-established. The galvanic excitability, however, remains a long time below its normal degree, so that at a certain period during recovery from paralysis the muscles may present a great diminution of galvanic with an increased faradic excitability.

The reason of the different reactions to the faradic and galvanic currents appears to be, as has been pointed out by Newman, that the paralysed muscles have lost the power of responding to currents of very short duration ; and since the faradic current is made up of successive shocks of momentary duration, the muscles do not respond to them.

The primary diminution of the electric excitability of the muscles runs a parallel course to the excitability of the nerve-fibres, and probably depends upon the degeneration of the terminal nerve-fibres within the muscle itself (Erb). The increase and qualitative changes of the galvanic excitability appear during the second week, this being the time during which the muscular substance is undergoing histological and chemical changes. The subsequent diminution of the galvanic excitability corresponds with the atrophy of the muscular fibres ; and the gradual return to the normal excitability with the regeneration of the muscle from the atrophy and cirrhosis which it had undergone.

Whenever, therefore, the "reaction of degeneration" presents itself it may be inferred that considerable anatomical changes must have taken place in the nerves and muscles, the exact nature of which may be deduced, with some degree of certainty, from the stage to which the electrical changes have advanced. These nutritive changes will be described under the section treating of the trophic neuroses.

The reactions obtained by the direct application of the galvanic current to the muscles may be formulated as follow :—

*Law of Normal Contraction.*

Weak currents .....	$\text{Ca S C}$			
Medium currents .....	$\text{Ca S C'}$	$\text{An S c}$	$\text{An O c}$	
Strong currents .....	$\text{Ca S T e}$	$\text{An S C}$	$\text{An O C}$	$\text{Ca O c}$

*Quantitative Changes (simple degeneration).*

SIMPLE INCREASE.				SIMPLE DECREASE.			
Weak currents .....	Ca S C'	An O C	1st degree.				2nd degree.
Medium currents ...	Ca S Te	An O C'		Ca S C	An S c	An O c	
Strong currents .....	Ca S Te	An O Te		Ca O C'		Ca S c	

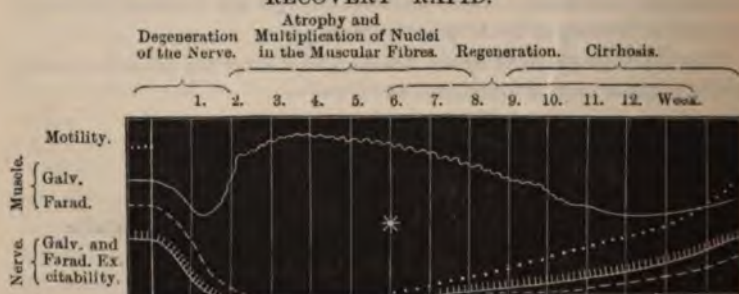
*Quantitative and Qualitative Changes—Reaction of Degeneration (Atrophic Degeneration).*

## Currents—

Weak.....	1. Stage of increase .....	Ca S C	An S Te	An O C	Ca O C'
Medium..	2. Stage of gradual decrease ...	Ca S c	An S Te		
Strong ...	3. Final stage prior to abolition		An S c		

The following diagrams (*Figs. 8, 9, 10*), borrowed from Erb,\* represent graphically the general relations of motility, excitability, and structural changes which are present in the different stages of paralysis. "The first thick vertical line or ordinate," says Erb, "indicates in all the drawings the attack of paralysis, the sudden cessation of motility (....); the period of return of motility is indicated by a star (\*). The succeeding ordinates represent intervals of one or more weeks, dating from the occurrence of the attack. The undulations in the line representing the galvanic excitability of the muscle indicate its qualitative changes. *Fig. 8*, for instance, exhibits the diminution of excitability that occurs, both in nerve and muscle, during the first week; the extinction of excitability of the nerves and of the faradic excitability of the muscle, the augmentation and qualitative change in the galvanic excitability of the muscle in the second week; and the return of the motility in the sixth week. In the eighth week it may be seen that the motility is restored to some extent, that the nerve has recovered its faradic and galvanic excitability, and that there is an increase and qualitative change in the galvanic excitability of the muscle, and so on." In the second degree of the reaction of degeneration the faradic and galvanic excitability of the nerve does not appear until the thirtieth week (*Fig. 9*); while in the third degree (*Fig. 10*) the excitability of the nerve never returns, but the galvanic excitability of the muscle only becomes finally abolished after a prolonged period, in some cases extending over a period of two years.

FIG. 8.  
RECOVERY RAPID.



\* Ziemssen's *Cyclopædia*, vol. xi., p. 436.



Fig. 9.

## RECOVERY SLOW.

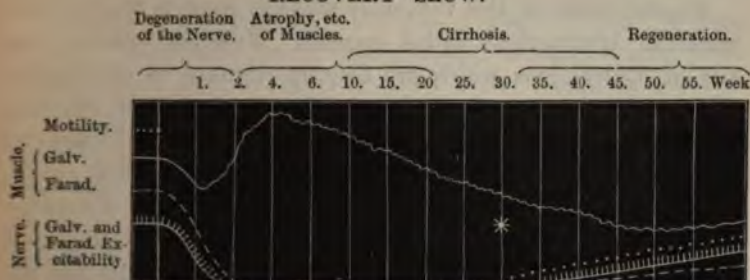
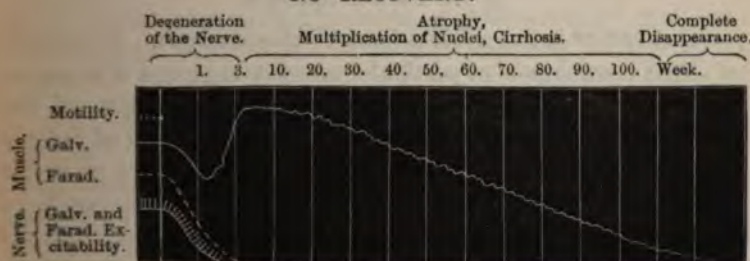


FIG. 10.

## NO RECOVERY.



The "reaction of degeneration" occurs in all paralyses of traumatic origin, accompanied by complete division or crushing of the nerves, in certain paralyses of rheumatic origin, especially in facial paralysis, and in paralysis from neuritis, and compression of the nerves from tumours and other causes. It also occurs in *saturnine* paralysis and in infantile paralysis, and in that which occurs in the course of or subsequent to acute diseases of the spinal cord.

*Mechanical Excitability.*—In some forms of paralysis the muscles exhibit an increased excitability to mechanical irritation. Paralysed muscles respond by a distinct slow and protracted contraction to mechanical stimuli, such as tapping with the tip of the finger or a blow with a light percussion hammer. The increased mechanical appears to be more or less connected with the increased galvanic excitability; but the augmentation of the former occurs at a somewhat later period than that of the latter. When once the mechanical excitability appears it increases rapidly, and is particularly well marked in muscles having a firm, bony support; it then gradually diminishes, and disappears in the course of the third or fourth month.

## HYPERKINESSES OF THE MUSCLES OF EXTERNAL RELATION.

*Hyperkinesis* of the voluntary muscles consists of abnormal muscular contractions called *spasms*. Muscular spasms consist of contractions which are disproportionate to the degree of external stimulus, or which arise in the absence of external stimulation as the result of pathological irritation. Spasmodic affections may be divided into two groups. In the first group, or *clonic* spasms, the affected muscles are in a state of rapidly alternating contractions and relaxations; while in the second group, or *tonic* spasms, the affected muscles are maintained for hours, or even days, in a state of persistent and equable contraction.

§ 68. *Clonic Spasms*.

(1) *Tremor* is the mildest form of clonic spasm. It consists of slight contractions of particular muscles or groups of muscles, by means of which a peculiar rhythmical oscillation of the limbs and trunk is produced. The higher degrees of tremor consist of more powerful contractions, which cause manifest trembling, or even distinct quivering of the limbs, such as is met with in *paralysis agitans*. *Fibrillary contractions* consist of alternate contractions and relaxations of individual bundles of muscular fibres, visible as wavy oscillations under the skin, but which do not give rise to any movement of the limb.

*Varieties*.—A very old division of tremor into two varieties has been revived by Charcot, Gubler, and others. The distinction was indeed made by Galen, and special attention was directed to it by Van Swieten. In the one variety the tremor persists during repose, and Van Swieten attributed this form to irritation, affecting the nervous centres in an intermittent and rhythmical manner. He regarded it as a convulsive phenomenon, and called it *tremor coactus*. In the second variety the tremor is exclusively shown during the execution of voluntary movements, and Van Swieten regarded it as a paralytic phenomenon, and called it *tremor a debilitate*. There can be no doubt that there are two varieties of tremor, the one persisting during repose, and the other only appearing when the patient makes a voluntary effort; but whether the



former is due to pathological irritation and the latter is a paralytic phenomenon is open to question.

Tremor is a symptom of various diseases of the nervous system. It appears locally as a symptom of neuritis and more extensively distributed as a symptom of certain central diseases of the nervous system; as disseminated sclerosis, paralysis agitans, and in chronic poisoning by alcohol, opium, lead, or mercury. It may sometimes occur as an independent disease, then it constitutes simple, essential, or idiopathic tremor. Simple uncomplicated tremor occurs generally in old age; but it is occasionally met with in young persons. Women appear to be more disposed to tremor than men, and it may occasionally occur as one of the manifestations of hysteria. Tremor occurs also as a symptom of exhausting diseases; such as typhoid fever, great bodily and mental exertion, excessive venery, onanism; and the rigor which ushers in acute disease may be regarded as a kind of tremor.

(2) *Convulsion*.—The next variety of clonic spasm is termed convulsion. Convulsive movements consist of energetic contractions and relaxations of particular muscles or groups of muscles, causing a rapid succession of vigorous movements, and giving rise to twitchings of the face, startings of the limbs, and movements of the head and body. If the majority of the muscles of the body are affected with alternating contractions and relaxations, causing extensive and irregular movements of the trunk and limbs, the condition is termed *general convulsions*, a condition which forms the most prominent feature of epilepsy, uræmia, eclampsia, and hysterical attacks.

### § 69. *Tonic Spasms.*

(1) *Cramp* is the simplest form of tonic spasm, and consists of a persistent painful contraction of a muscle, or of a group of muscles. Cramp of the calf of the leg is the most common variety of this affection when it is limited to a single muscle. It frequently comes on during sleep, more especially after the muscles of the calf have been strained the previous day by prolonged walking or by dancing. Very painful and troublesome cramps of the abdominal muscles, and of those of the lower extremities, occur in severe cases of summer diarrhoea; but these

cramps assume their highest severity and importance in Asiatic cholera. It is doubtful whether the cramps of cholera are caused by reflex irritation or by nutritive changes in the muscles and nerves induced by the disease.

In tetanus the majority of the muscles of the body are affected with cramps which recur in paroxysms of longer or shorter duration.

A peculiar modification of cramp is met with in catalepsy. The muscles in this affection are in a condition of moderate contraction; but the resistance they offer may be readily overcome by passive movements, so that the limbs may be made to assume constrained positions, which they retain. From the manner in which the limbs can be moulded into various positions, this condition has been called *flexibilitas cerea*.

(2) *Muscular tension* is a state of moderate contraction of certain muscles or groups of muscles, which occurs when they are stretched either by passive movements or by a voluntary contraction of their antagonists. This condition is always associated with a certain degree of loss of voluntary power over the affected muscles.

(3) *Contracture* is meant to express any persistent shortening of a muscle, whereby its points of origin and insertion are permanently approximated. Muscular contractures are of various kinds. In the first group the contracted condition is due to primary changes in the muscular substance itself, hence it may be called *myopathic contracture*. The muscular changes consist of induration of the connective tissue, accompanied by fatty degeneration and subsequent atrophy of the muscular fibres. The structural changes in the muscle are indeed due to a sub-acute inflammation of its substance, a myositis which from the increased growth and subsequent shrinking of the connective tissue may be called *muscular cirrhosis*, from its analogy with cirrhosis of the liver. This process may result from direct injury to the muscle, or from acute rheumatism, syphilis, lead poisoning, and various other causes. Another form of permanent muscular contraction may be called *paralytic* or *secondary contracture*. When a certain group of muscles is paralysed, their antagonists have their ends approximated; and after a time they become accommodated to their new positions and be-



come permanently contracted. At other times a paralysed limb assumes a certain position in accordance with the law of gravitation, such as the position of talipes equino-varus assumed by the foot in infantile paralysis, and in this way the points of attachment of certain groups of muscles are approximated and then become permanently shortened. In long-standing disease of the joints, when the bones have to assume false positions with respect to one another, the extremities of some of the muscles are unduly approximated, and then become permanently contracted.

But the form of muscular contraction, which more immediately concerns us at present, is that which is caused directly by abnormal innervation, and which may therefore be called *primary neuropathic contracture*. The muscles in this, as in the other forms of contracture, become persistently rigid and shortened, and thus give rise to various deformities. It is always associated with a certain amount of paralysis. The rigidity of the muscles usually disappears during sleep and gradually returns on awakening, and it is almost always increased by voluntary movement.

§ 70. *Concomitant Symptoms*.—Spasms are either accompanied or followed by various other phenomena, which deserve mention. Other motor disturbances are frequently present, generally in the form of diminution or loss of voluntary power over the affected muscles. During the height of a convulsion associated movements sometimes occur in more or less numerous groups of muscles, in consequence of irradiation of the irritation into the various intercommunicating paths.

Sensory disturbances are frequently associated with spasmodic affections. A sensation of fatigue and exhaustion is often felt in the muscles after the cessation of the cramps; and the muscles also feel sore and tender to pressure just as occurs in healthy muscles subjected to undue strain. Pain is often present in the contracted muscles; while spasm due to an affection of a mixed nerve is frequently accompanied by severe neuralgic and excentric pains, formication, numbness, or well marked anæsthesia.

Vaso-motor and secretory disturbances are frequently wanting,

but occasionally localised paleness, redness, or cyanosis of the surface is observed. Profuse perspirations occur both in epilepsy and tetanus, and sweating on one side of the body has been observed in unilateral epilepsy; and after severe attacks of convulsions there is frequently an abundant flow of clear limp urine (*urina spastica*).

The trophic disturbances are usually insignificant. Muscles affected with the severest spasms may exhibit neither hypertrophy nor atrophy; but the latter condition is the more frequent.

Psychical disturbances are not unfrequently associated with spasms; more especially in epilepsy and hysteria. It is very remarkable how little the general health suffers from severe spasmodic affections.

§ 71. *The Consecutive Symptoms or Sequelæ* of spasm are for the most part mechanical, and result from the undue muscular contraction. The most important of these are impairment of the mobility of joints, abnormal positions of the head and limbs, curvatures of the spinal column, alterations of the articular extremities of the bones, displacements and subluxations of the joints, disturbances in the functions of special organs, as strabismus, and difficulty of breathing or swallowing, urination and defæcation.

The state of the electric contractility of the affected muscles and nerves varies greatly, being sometimes normal, at other times diminished, and at other times increased.

§ 72. *Pressure Points* are frequently observed in spasmodic affections. Pressure upon certain points puts a stop at times to the convulsion when present, and consequently these points may be called *motor arresting pressure points*; whilst in other cases the convulsions are brought on by pressure made on particular points; hence these may be called *motor exciting pressure points*. Pressure points of the first kind have been particularly observed in the case of facial spasm, and they correspond, like the painful points in neuralgia, to the various branches of the trigeminus, and they are not unfrequently sensitive to pressure. Similar pressure points have also been observed in other forms of convulsion than those affecting the face.



§ 73. *Theory of Hyperkinesis.*

Schiff was the first to observe that tremor is frequently seen in muscles which are severed from their connections with the voluntary nervous centres. This phenomenon is best seen in the muscles of the tongue of the dog after section of the hypoglossus. Vibratory movements of the bundles of muscular fibres may be seen through the mucous membrane; and when one only of the nerves has been cut, the tremor is confined to the paralysed side. The tongue is not moved as a whole by these contractions. A few bundles enter into contraction at a time, and when these relax other bundles contract, and thus the organ is maintained in a constant state of tremor. In rabbits there is continual oscillation of the whiskers after section of the facial; and in birds, trembling of the iris, which is provided with striated fibres, after section of the motor oculi. Similar trembling occurs in the muscles of the extremities when their nerves have been separated from the spinal centres. The trembling does not begin until some days after section of the nerves, and it gradually reaches its maximum towards the end of the first week, and may then continue months or even years. During this time the peripheral portion of the nerve is undergoing centrifugal degeneration; and as this degeneration induces consecutive changes in the muscles, it is very probable that the tremor is a symptom of the structural alteration which the muscles have undergone, or that it is due to pathological irritation of the motor terminal plates in the interior of the muscles. Trembling is a marked symptom not only of affections of the peripheral nerves, but also of diseases of the pyramidal tract; in one word, it occurs whenever the influence of the voluntary centres over the muscles is greatly weakened or abolished, and consequently the opinion of Romberg is not without justification that tremor may be said "to form the bridge from spasm to paralysis." A tonic spasm of a muscle is caused by the fusion of rapidly recurring simple spasms into an apparently smooth continuous contraction. When the resistance to conduction is greatly increased the successive nervous shocks do not recur with sufficient rapidity to produce a continuous contraction, but a series of alternating contractions and relaxations occur, which is the essential condition underlying tremor.

Another theory of the nature of tremor has also found acceptance with some modern pathologists. The various muscular adjustments of the body are regulated, as already stated, both by the cerebellum and cerebrum; the former presiding over the tonic, and the latter over the clonic muscular contractions. In conformity with this theory, tremor results from a loss of balance between the actions of the two great centres of innervation, being more especially due to the tonic action of the cerebellum when the action of the cerebrum is enfeebled (Jackson). Contracture, again, is supposed to be caused by the complete abolition of the cerebral influence, the action of the cerebellum being still maintained in a normal condition. Reflex spasms are caused by irritation of, or excess of

irritability of the reflex arc in any part of its course, either of the afferent or efferent fibres, or of the grey substance of the spinal cord itself.

The theory of general convulsions cannot be satisfactorily discussed at present. Schröder Van der Kolk first suggested that general convulsions were due to irritation of the upper portion of the medulla oblongata; and Nothnagel has recently shown that they may be induced by irritation of a limited portion of the floor of the fourth ventricle, which he has consequently named the convulsive centre. Various experimental and pathological facts, however, favour the idea that general convulsions, accompanied by unconsciousness, are determined by a discharge from the cortex of the brain. The cortical discharge may, of course, be induced not only directly by local irritation, but also indirectly by irritation of any portion of the centripetal apparatus. Dr. Hughling Jackson, who was the first to enunciate this theory, also thinks that the tonic contractions of tetanus are caused by a nervous discharge from the cortex of the cerebellum.

#### ELEMENTARY AFFECTIONS OF THE REFLEX MECHANISM OF THE MUSCLES OF EXTERNAL RELATION.

The disturbances which occur in the reflex mechanism situated in different parts of the nervous system are almost infinitely numerous. A very considerable number of the spasmodic affections already described belong to this category, and in most forms of paralysis reflex action is decidedly implicated; while the reflex disorders of the vaso-motor mechanisms, which will be subsequently described, are exceedingly important and diversified in character. It will, however, be useful to mention here a few of the disturbances which occur in the more common and simple reflexes of the spinal cord. The reflex system of the spinal cord consists of a series of nerve loops, the afferent portions of which pass in through the posterior roots of the nerves, and become connected, through a mechanism of cells and fibres, with an efferent fibre or fibres, which pass out with the anterior roots of the nerves, to be conducted to the muscles.

*Time required for Reflex Actions.*—In a reflex act an impression is made upon afferent fibres, which convey the impulses to the spinal cord, where they are reflected through efferent fibres to a muscle or muscles. Such a process as this requires an interval of time to elapse between its initiation and completion, and the period which intervenes between the application of the stimulus and the beginning of the contraction which ensues is called the latent period. The length of this latent



period is readily calculated theoretically, although considerable practical difficulties may present themselves. This period is made up of three intervals :—

1. *The first interval is occupied in conducting impulses along afferent and efferent fibres.*—Conduction through a metre in length of a nerve takes place in  $\cdot 03$  of a second; and if  $x$  be equal to the conjoined length, in metres, of the afferent and efferent fibres engaged in a reflex act, the time occupied by conduction through them will be equal to  $\cdot 03x$ .

2. The second interval consists of the time consumed in the central operations of the reflex act, and the length of which Exner has estimated at  $\cdot 055$  of a second.

3. The third portion of the interval consists of the time occupied in setting up molecular changes in the muscle, unaccompanied by any visible alteration in its form, this period occupying on an average about  $\cdot 01$  of a second.

By adding the estimated lengths of these three portions of time together, we obtain the length of the interval which elapses between the application of a stimulus and the occurrence of the resulting reflex contraction, the formula being  $\cdot 03x + \cdot 055 + \cdot 01$ . It ought to be remembered, however, that the time for any reflex act varies greatly within healthy limits, according to the strength of the stimulus employed, and according as the nutrition of the cord and nerves, and indeed of the cephalic ganglia, is active or in an exhausted condition. When once the muscular contraction begins the shortening of the muscle up to a maximum occupies about  $\cdot 04$ , and its return to its former length  $\cdot 05$  of a second.

§ 74. *Disease of the reflex mechanism* may declare itself by way of excessive reaction, constituting *reflex hyperkinesis*, or by diminution or loss of reaction, constituting *reflex akinesis*.

*Reflex Hyperkinesis* is caused by diseases which increase the irritability and consequently diminish the specific resistance of the reflex arc in any part of its course; those which arrest conduction through the fibres of the pyramidal tract; and those which increase the irritability of the muscular fibres themselves.

*Reflex Akinesis*, on the other hand, is caused by diseases which diminish or abolish the irritability, and consequently increase the specific resistance of the reflex arc in any part of its course, those which increase the cerebro-spinal discharges passing through the pyramidal tract, and those which diminish or abolish the irritability of the muscular fibres themselves.

*Localisation of the Lesion.*—The irritability of the reflex arc may be increased, diminished, or abolished by disease affecting—

(1) The afferent fibres of the arc from their peripheral origin until they pass through the posterior roots of the nerves (*Fig. 11, p*) to end in the grey substance of the posterior horns. When either the afferent fibres or their peripheral or central terminations are affected, the reflex symptoms are usually accompanied by sensory phenomena.

(2) The efferent fibres of the reflex arc, at their central origin in the ganglion-cells of the anterior horns, in their passage through the anterior root zones, anterior roots (*Fig. 11, a a*), and peripheral nerves; and finally, in their peripheral terminations in the individual muscular fibres. In disease of the efferent fibres, or of their central or peripheral terminations, the reflex symptoms are usually accompanied by voluntary motor disorders.

(3) The grey substance of the reflex mechanism (*Fig. 11, P, A*).

(4) The conducting path—the pyramidal tract—which connects the spinal centres with the higher cerebral centres (*Fig. 11, p t*).

(5) The muscular fibres themselves.

*Varieties of Reflex Actions.*—The reflex actions may be subdivided into two forms—the superficial, and the deep.

§ 75. *The Superficial Reflexes* are excited by stimulation of the skin and accessible mucous membranes.

The tests employed for estimating the various degrees of these reflexes are tickling, pricking, pinching, or gently scratching the surface, or the application of the faradic current to the surface by means of dry electrodes or of the faradic brush.

A series of reflex actions may be obtained from the normal spinal cord, which, as Dr. Gowers\* has shown, are of the utmost importance in the diagnosis of spinal affections.

The following superficial reflexes may be distinguished :—

1. *The reflex of the sole* of the foot, which depends upon the integrity of the reflex arc through the lower end of the cord (*conus medullaris*).

2. *The gluteal reflex*, consisting of contraction of the gluteal muscles, induced by stimulating the skin over the buttock, and depending upon the integrity of the arc through the fourth or fifth lumbar nerves.

\*The diagnosis of diseases of the spinal cord, by Dr. W. R. Gowers.—*The Medical Times and Gazette*, Vol. II., p. 526.



3. *The cremasteric reflex*, by which the testicle is drawn up when the skin on the inner side of the thigh is stimulated, and demanding the integrity of the first and second pair of lumbar nerves.

4. *The abdominal reflex*, consisting of a contraction of the abdominal muscles, chiefly the rectus, caused by stroking the skin on the side of the abdomen from the edge of the ribs downwards, and requiring the integrity of the arc through the nerves from the eighth to the twelfth dorsal nerves.

5. *The epigastric reflex* producing a dimpling of the epigastrium on the side stimulated. It is induced by stimulation of the side of the chest in the sixth, fifth, and sometimes fourth intercostal spaces. This dimpling probably depends upon contraction of the highest fibres of the rectus abdominis, and its presence requires the integrity of the cord from the fourth to the sixth or seventh pairs of dorsal nerves.

6. *The erector spinæ reflex*, consisting of a local contraction of these muscles, caused by stimulation of the skin along their edge from the angle of the scapula to the iliac crest, and demanding the integrity of the reflex arcs in the dorsal region of the spinal cord.

7. *The scapular reflex*, consisting of a contraction of some, or nearly all, of the scapular muscles according to its degree, and demanding the integrity of the cord at the level of the upper two or three dorsal and lower two or three cervical nerves.

8. *The palmar reflex* consists of a contraction of the flexors of the fingers induced by tickling the palm of the hand. It requires the integrity of the reflex arcs through the greater part of the cervical enlargement. This reflex is not readily induced during waking hours and consequent cerebral activity, probably because the hand is much more under cerebral influence than the foot. During sleep, however, and in young infants, when the cerebral influence is suspended, or not yet fully established, this reflex is as readily induced as the reflex of the sole of the foot.

9. *Cranial reflexes*.—The chief reflexes of the cranial nerves are the contraction of the palatal muscles caused by irritation of the fauces; the sneezing caused by irritation of the mucous membrane of the nose; the cough caused by irritation of the mucous membrane of the larynx; the closure of the eyelids caused by irritation of the conjunctiva; and the reflex contraction of the iris caused by light.

Some of these reflexes are absent in healthy individuals, more especially the reflexes of the back and abdomen, so that the diminution or absence of some of them must not be taken as a sure sign of disease. Their presence, however, is a proof that the respective paths through the cord are not seriously interrupted.

Increase of these reflex reactions indicates that the irritability of the respective arcs is increased in some portion of their course, or that the inhibitory influence of the cerebrum is withdrawn. When a frog is poisoned with strychnia, a slight touch on any part of the skin may cause convulsions of the whole body, due, as Dr. Ringer has shown, to diminution of

the specific resistance of the grey substance. A similar increase of the reactions obtained by cutaneous stimulation occurs in strychnia poisoning in man. When the brain of the frog is removed reflex actions are developed to a much greater degree than in the entire animal; but if the optic lobes be stimulated by putting a crystal of sodium chloride upon them the activity of the reflex actions becomes again diminished. That withdrawal of cerebral influence increases the reflex activity of the spinal cord in man is shown by the facts that the reflexes are very active during sleep and in childhood.

The condition of the cutaneous reflexes in cerebral paralysis, however, appears to be an exception to this rule, inasmuch as they become greatly diminished or abolished on the paralysed side. It has been shown by Rosenbach that the abdominal reflexes, and by Jastrowitz that the cremasteric reflexes, are abolished or diminished on the paralysed side in cases of disease of one cerebral hemisphere. These curious facts may probably be due to paralysis, or loss of tone of the muscular fibres distributed to the skin, which may be followed by so much cutaneous flaccidity as to prevent the peripheral termination of the afferent fibres of the reflex arcs from being duly exposed to the irritation of tickling and other cutaneous stimulants. But, whatever may be the explanation, there can be no doubt that these phenomena are only apparent and not real exceptions to the general law, that diminution of cerebral influence, other things being equal, increases the reflex activity of the cord.

### § 76. *The Deep Reflexes.*

The deep reflexes consist of muscular contractions, evoked by striking the muscles themselves, or stretching their tendons, or even, under certain circumstances, by tapping certain parts of the periosteum, and probably some of the fasciæ. The reflex muscular contraction obtained under certain conditions on striking a smart blow across a muscle must not be confounded with the idio-muscular contractility already described, which is always in excess when the nutrition of the muscle is in an exhausted condition. But as the mechanical reflex contractility of the muscle is subject to the same laws as the tendinous reflex, the former does not demand a separate examination.

§ 77. *Tendinous Reflexes.*—Of the phenomena which have been grouped under the name of *tendon reflexes*, by far the best known are those which have been called respectively “knee-phenomenon” and “foot-phenomenon,” by Westphal, and patellar-tendon and Achilles-tendon reflex, by Erb. The former



has also been called *knee reflex*, and the latter *ankle reflex*, or *ankle clonus*.

*Patellar-tendon Reflex.*—If a man in health sits with one leg crossed upon the other, and the ligamentum patellæ be then smartly struck immediately below the knee-cap, the extensor muscles in front of the thigh become suddenly contracted, causing the foot to be jerked forwards to a variable extent, according to the extent of the contraction.

The blow is usually delivered by the inner edge of the hand of the operator; but an ordinary stethoscope, held loosely by the small end while the blow is struck with the edge of the ear-piece, is a convenient instrument for the purpose; while, in cases which require great delicacy in the application of the test, it is desirable to use a Winterich percussion hammer for the purpose. It is also desirable to uncover the knee that the blow may be delivered on the bare skin.

The usual sitting posture, with the legs crossed so that the back of one knee-joint rests on the front of the other, is the position generally adopted for applying the test; but any position will suffice which renders the tendon tense and leaves the leg free to move, and indicate the contraction. Sitting on an elevated seat, the legs hanging freely, is also a convenient position; and in stout people, who cannot readily cross one leg over the other in a sitting posture, the operator may pass his hand beneath the patient's thigh just above the knee joint, and, grasping the opposite knee, support the extremity to be examined by his forearm (Gowers).

The reaction is most energetic when the blow is struck a little below but very near to the patella; and it may be necessary to make repeated trials before the most sensitive spot is discovered. Although the most sensitive spot is over the tendon immediately below the patella, the contraction may also be excited, more especially when the reaction is lively, by a blow on the tendon above the patella, or even by a blow on the muscle.

The strength of the contraction varies greatly within the limits of health in different individuals, and even in the same individual at different times. With myself this reflex is at times very lively; while at other times it is not readily elicited, although the phenomenon is always present to some extent. The conditions which determine these variations are not clearly traceable. In the case of a medical friend of mine, who enjoys the most perfect health, the patellar reflex is entirely absent. Both of us have applied the test many times during the last eighteen months, and with every known precaution against failure, but never succeeded in obtaining the slightest trace of a contraction of the quadriceps. Out of 1,409 healthy individuals examined by Berger the patellar reflex was absent in 22, or 1.56 per cent.\*

\* Centralbl für Nerven heilkunde, No. 4. 1879.

The mechanism by which this reaction is produced has been much discussed of late. It is conceivable that the contraction of the quadriceps is of the nature of idio-muscular contractility, and induced by the direct irritation of the muscular fibres which their sudden stretching causes; or it may be of the nature of a true spinal reflex. The first supposition has been maintained by Westphal; but the second opinion, originally advanced by Erb, is now almost universally adopted.

If this reaction is a true spinal reflex, it must conform to the laws which regulate the genesis and transmission of other reflex acts, and must consequently possess the following characteristics:—

1. The reaction must take its origin from stimulation of afferent fibres.
2. An interval must intervene between the instant at which the blow is delivered on the tendon and the commencement of the contraction of the quadriceps, corresponding to the time occupied in the transmission of other reflex impulses.
3. The reaction ought to be diminished or abolished by all injuries and diseases which diminish or abolish the irritability of any portion of the reflex arc concerned in its production; and conversely it ought to be augmented by those lesions which increase the irritability of any portion of that arc.

1. The first requirement laid down is that the reflex act must take its origin from stimulation of afferent fibres. Now if the knee phenomenon be a true reflex it must conform to this requirement, and the origin of the afferent fibres concerned must, if possible, be determined. The afferent fibres of the knee phenomenon might spring from the skin over the tendon, from the tendon itself, or from the muscles. That the cutaneous nerves are not concerned in the reaction has been amply proved by Westphal and others. Westphal could not induce any contraction in the muscle by pinching the skin over the tendon, or by striking it with a percussion hammer after being raised up in a fold so that it could be struck without acting on the underlying tendon; and the reaction is readily obtained in animals by striking the bare tendon after the skin has been cut.

It is not so easy to determine whether the afferent fibres start from the tendon or from the muscle. In favour of the former view it may be stated that Sachs has demonstrated the existence of nerve fibres in the tendon of the quadriceps, these being especially abundant at the point of junction of the tendon with the muscles. Without, therefore, denying that the afferent fibres starting from the muscle are concerned in the reaction, it is exceedingly probable that afferent fibres exist in the tendon, stimulation of which will occasion the phenomenon.

2. Let us now see if the patellar-tendon reflex conforms to the second requirement of a reflex act. We have already found that if  $x$  is equal to the combined lengths of the afferent and efferent fibres of a reflex arc, the time which intervenes between an impression and the resulting contraction is equal to  $0.3x + 0.055 + 0.01$  of a second. Now, suppose that, with Dr. Gowers, we assume that the length of the afferent and



efferent fibres of the knee reflex arc—that is, the length from the tendon to the spinal cord at the level of the origin of the sixth lumbar nerve and back again to the middle of the rectus-muscle—is equal to a metre and a half (which is rather too much), then our formula is  $\cdot 03 \times 1\cdot 5 + \cdot 055 + \cdot 01 = \cdot 11$ , or about  $\frac{1}{9}$  of a second.

The graphic method has been employed by several observers in order to determine whether the interval between the blow on the tendon and the contraction of the quadriceps corresponds to the conclusion derived from the reflex formula. There are considerable discrepancies between the results obtained by different observers. Burckhardt and Tschirjew, for instance, have found the interval between the blow and the contraction not to exceed  $\cdot 04$  to  $\cdot 05$  of a second; and Brissaud and Franck, who conducted observations on this point under the direction of Charcot,\* found the length of the interval to be  $\cdot 04$  in health, and  $\cdot 036$  in lateral sclerosis where the reaction is exaggerated; while, on the other hand, Dr. Gowers found the length of the interval to average  $\cdot 10$  or  $\cdot 11$  of a second, an estimate which corresponds exactly with the conclusion derived from the reflex formula. Notwithstanding the discrepancies between the estimates of different observers, the results obtained on the whole favour the idea that the contraction of the quadriceps is a true spinal reflex. In the very careful graphic tracings given by Dr. Gowers, a slight initial elevation is observed to be interposed between the blow on the tendon and the large elevation caused by the energetic contraction of the muscle. This initial elevation begins  $\cdot 05$  of a second after the blow is delivered, and Dr. Gowers attributes it to a slight primary contraction of the muscle caused by the direct mechanical irritation of the muscular fibres produced by their sudden stretching on the tendon being struck.

3. The third condition mentioned is very complex, inasmuch as a lesion, whether it increase or diminish the irritability of the reflex arc, may occupy so many different positions. Notwithstanding the complexity of the problem, a considerable number of facts have now been accumulated, both by physiologists and pathologists, which render the proof in this respect almost as cogent as could be desired.

Fürbinger and Schultze found that the knee-reflex exists in animals, and that it was abolished on the destruction of the spinal cord. Tschirjew also found that destruction of the portion of the spinal cord of the hare, opposite the fifth and sixth lumbar vertebræ, and from which the greater portion of the anterior crural nerve springs, as well as section of the posterior roots of the sixth lumbar pair of nerves, immediately arrested the knee-reflex, while section of the cord above and below this point did not exert any manifest influence on its production. These observations have since been confirmed by Senator. It ought to be mentioned that Burckhardt states that in animals, although section of the anterior crural nerve arrests the knee-reflex, yet it is not arrested by section of the

\* *Le Progrès Médical*, 13 Mars, 1880, p. 204.

spinal roots, and hence he concludes that the phenomenon is a reflex from the ganglia of the posterior-roots. This conclusion is, however, contrary to well ascertained pathological facts, and the experiment upon which it is founded is, moreover, contradicted by the positive results obtained by Tschirjew. Turning now to pathological facts, the evidence in favour of the reflex nature of this reaction is no less convincing; and, indeed, the pathological facts which prove the reflex origin of the knee-phenomenon are those which render it such an invaluable sign of the condition of that portion of the spinal cord from which the anterior crural nerve arises.

§ 78. *Diseased conditions under which the Patellar-tendon Reflex is diminished or abolished.*—(1) The patellar-tendon reflex is absent in most cases of locomotor ataxy, and the diagnostic value of this symptom is greatly enhanced by the fact that the phenomenon usually disappears at a very early period of the affection. It is now well known that the morbid lesion in locomotor ataxy is situated in the posterior root-zones (*Fig. 11, pr*) and posterior roots, and consequently the absence of the knee-reflex is usually associated with lightning pains and various other sensory disturbances. It is probable, therefore, that conduction through the patellar-tendon reflex arc is arrested in this affection in the afferent fibres towards their insertion into the posterior grey horns of the cord, the exact locality being the internal bundle of the posterior root, or what Charcot calls the *inner radicular fasciculus* (*Fig. 11, p'r*). Senator, however, found that in the hare, although section of the cord opposite the fifth and sixth lumbar vertebræ arrested the reflex, yet neither section of the posterior columns, posterior horns, nor anterior horns in this locality had any influence upon it. On the other hand, the reflex was arrested by section of the lateral columns and the adjoining grey substance. It is therefore probable that the afferent fibres of this reflex arc pass into the posterior grey horn in the hare in the external, instead of the internal bundle of the posterior roots as in man. The frequency with which the knee phenomenon is absent in cases of locomotor ataxy affords a strong corroboration of the theory of the reflex origin of the phenomenon; and another curious observation has been made by Erb which gives further confirmation to this opinion, if any further were needed. He has shown that in those cases in which the patellar-tendon reflex is completely



abolished a slight contraction of the quadriceps femoris may still be obtained by dealing a smart blow across the middle of the muscle with the edge of the extended hand, showing that the mechanical contractility of the muscle is retained, although the reflex is abolished.

FIG. 11.

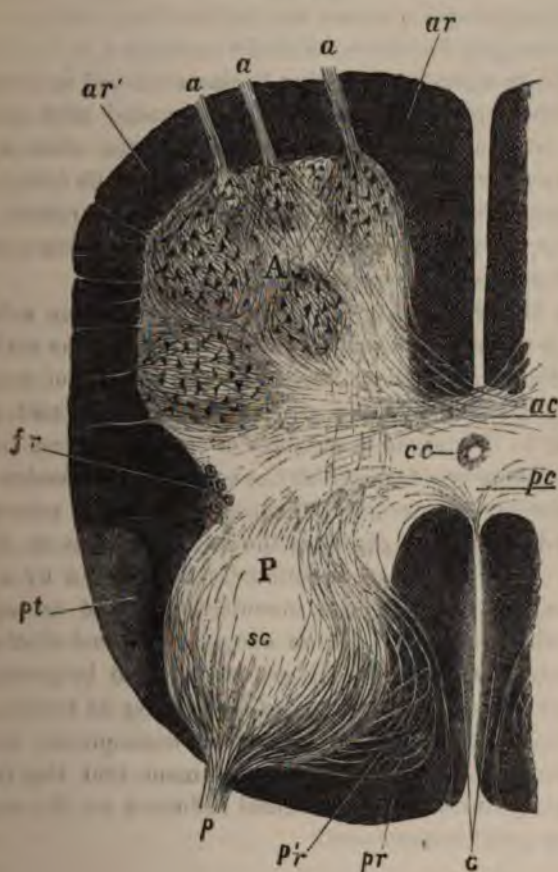


FIG. 11. *Section of Spinal Cord from the middle of the Lumbar Enlargement.*—A, P, Anterior and Posterior Grey Cornua respectively; sg, Substantia Gelatinosa; cc, Central Canal; ac, pc, Anterior and Posterior Commissures respectively; G, Column of Goll; pr, Posterior Root Zone; p, Posterior Root; p'r, Internal Radicular Fasciculus; a, a, a, Anterior Roots; ar, ar', Anterior Root Zone; fr, Formatio Reticularis; pt, Pyramidal Tract.

The posterior roots of the nerves are also not unfrequently implicated in cases of spinal meningitis, and in a case of the kind under my care at present, attended by shooting pains but without distinct diminution of any form of sensibility, both the superficial and deep reflexes are completely absent in the lower extremities; while the absence of muscular atrophy, of any decided loss of faradic and galvanic contractility, and of the reaction of degeneration, shows that neither the anterior roots nor the anterior grey horns are decidedly implicated in the disease.

(2) The patellar-tendon reflex is also abolished in diseases of the anterior grey horns (*Fig. 11, A*), attended with muscular atrophy, and no doubt also in disease of the efferent fibres of the anterior roots (*Fig. 11, a, a, a*) and of the crural nerve. This reflex is absent, for instance, in infantile paralysis, implicating the quadriceps muscle, and in the more or less advanced stages of pseudo-hypertrophic paralysis.

(3) It is not necessary that disease of the grey substance should be a permanent one in order to diminish the activity of the patellar-tendon reflex. Large doses of bromide of potassium lower the irritability of the grey substance of the cord, and so, according to Berger, do large doses of opium, and free administration of these agents diminish the activity of the tendon-reflex.

(4) Increase of cerebral influence on the grey substance of the spinal cord should diminish the activity of this as of other reflexes. This reflex can undoubtedly be arrested by a voluntary effort; and during the convulsive stage of an epileptic attack, which is caused by an excessive cortical discharge, I have found that not the slightest effect could be produced on the contractions of the quadriceps by striking its tendon. This observation is of course of very little consequence, but it is mentioned because the converse statement that the reflex is increased by diminution of cerebral influence on the cord is of such very great importance.

§ 79. *Diseased conditions in which the Patellar-tendon Reflex is exaggerated.*—(1) As already remarked, whatever increases the irritability of the reflex loop concerned in the reaction, or diminishes the cerebral influence on the spinal cord, increases the activity of this reflex. It is probable that irrita-



tion of the peripheral terminations of both the afferent and efferent nerve fibres will increase the activity of the reaction, although no crucial examples of the kind have as yet been described. When, however, the irritability of the muscular fibres themselves is increased, as in phthisis and other exhausting diseases, the tendinous reflexes are much more readily induced than in health.

(2) Irritation of the posterior roots of the nerves probably also increases the tendinous reflexes; but direct evidence upon this point is wanting as yet. We have seen that the patellar-tendon reflex is usually absent in an early stage of locomotor ataxy; but it is worth while to observe closely whether the abolition of the reflex may not be preceded by a transitory period of exaggerated reaction, just as anæsthesia is often preceded by hyperæsthesia. In a case under my care at present there are distinct ataxic symptoms along with excess of the patellar-tendon reaction; but it would be hazardous as yet to declare that the case is one of true locomotor ataxy. There can be no doubt that this reflex does occasionally persist in genuine cases of locomotor ataxy, although I am not aware that it has ever been described as exaggerated.

(3) Increased irritability of the grey substance of the spinal cord is attended with increase of the patellar-tendon reflex; hence the activity of the reaction is increased by the administration of strychnia. It is also very probable that it may be found increased in the early stages of acute diseases affecting the grey substance of the cord, such as tetanus, hydrophobia, and acute central myelitis; but I have not had an opportunity of testing this supposition since my attention was drawn to it.

(4) By far the most important condition, under which excess of patellar-tendon reflex occurs, is that in which the cerebral influence is withdrawn from the spinal cord by disease of some portion of the pyramidal tract. The effect on this reaction is the same, no matter where the disease of the pyramidal tract is situated, whether at the origin of these fibres in the cortex of the brain, in their passage through the corona radiata, internal capsule, pons, medulla oblongata, or in the spinal cord itself (*Fig. 11, pt.*). The only condition necessary for the subsequent development of the exaggerated reaction is, that con-

duction through the fibres of the tract be interrupted in any part of their course. But, although withdrawal of the cerebral influence from the lumbar portion of the cord is the necessary antecedent of the exaggerated reaction of the patellar-tendon reflex, it would appear that some other changes must take place before its activity is fully developed. When the fibres of the pyramidal tract are ruptured in the internal capsule by a sudden effusion of blood into the lenticular nucleus, hemiplegia of the opposite side results; yet the exaggeration of the patellar-tendon reflex does not become manifest until from eight to fourteen days after the attack. It is now well known that the fibres of the pyramidal tract below the point of rupture undergo a descending degeneration, a process which occupies a period of from one to two months. Closely associated with the completion of this process, at least with respect to time, is the occurrence of increased *tension* in the paralysed muscles, giving rise to the *contracture* already described, and constituting what has been described as the *late rigidity* of hemiplegic limbs. Without waiting to enquire into the cause of *late rigidity* it is probable that when it is once established the peripheral terminations of both the afferent and efferent fibres are maintained in a constant state of irritation by the continuous muscular tension, and that this condition adds to the irritability of the reflex arc already in excess from arrest of the inhibitory action of the brain on the cord. One forcible objection may be urged against this view. Although exaggeration of the patellar-tendon reflex and increased muscular tension are undoubtedly closely associated, yet the former manifests itself unmistakably in hemiplegic limbs in from eight to fourteen days, while the latter is not established in less than from one to two months from the date of the apoplectic attack. Therefore neither the arrest of cerebral influence, nor the occurrence of muscular tension, nor both combined, will fully account for the presence of exaggerated patellar-tendon reflex in disease of the pyramidal tract; hence some factor in its production must for the present remain undetermined. The great fact which concerns us is the invariable presence of exaggerated patellar-tendon reflex in diseases of the pyramidal tract, provided that the spinal centre of the anterior crural



nerve be severed from the cortex, and the occurrence of the phenomenon be not prevented by disease of the reflex arc itself. When disease of the pyramidal tracts becomes associated with locomotor ataxy, for instance, the patellar-tendon reflex remains as a rule absent (Westphal). I have found it also completely abolished in both legs in a case of meningo-myelitis in which the presence of paralysis and muscular tension showed that the pyramidal tracts were diseased, while the absence of atrophy and the maintenance of the faradic and galvanic contractility in both muscles and nerves showed that the grey matter was not affected.

When the reflex is exaggerated the slightest tap on the tendon induces an energetic contraction of the quadriceps muscle, and by repeating the blows in quick succession the contractions occur so close upon each other, and the interval of relaxation is so reduced, that the muscle is maintained in a state of almost tetanic contraction. Dr. Gowers has observed in cases where the reflex is much exaggerated that a single blow is followed by a succession of contractions and relaxations similar to the ankle clonus, to be hereafter described, with this important difference, that, whereas six or seven contractions occur within a second of time in the latter, only about two and a half of the quadriceps take place in that time.

Hitherto I have only spoken of the patellar-tendon reflex, but similar reactions may be obtained by striking the stretched tendons of other muscles. But inasmuch as no other muscle besides the quadriceps femoris, not even the triceps in the upper extremity, lends itself so readily to the demonstration of this reaction in health, so in no other muscle does the absence of the reaction afford such a valuable diagnostic sign. Exaggeration of this reaction in other muscles is, however, as significant of disease as when it occurs in the quadriceps. In disease of the pyramidal tract, for instance, in which the spinal centres of the upper extremity are cut off from cerebral influence, contractions may be obtained from the triceps by striking its tendon when the forearm is flexed at right angles to the arm, and from the muscles which move the fingers, by striking their tendons with the edge of the large end of the stethoscope as they become superficial at the wrist and back of the hand.

§ 80.—*Ankle Clonus, Achilles-Tendon Reflex, or Ankle Reflex*, consists of a rhythmical clonic spasm, which can be obtained under certain circumstances at the ankle-joint. Unlike the patellar-tendon reflex, the ankle reflex is not a phenomenon which can be readily induced in typically healthy individuals. The conditions necessary for the production of this reaction can, however, by a little preparation, be cultivated in most healthy people. If, for instance, a healthy individual sit on the edge of a chair, with the leg forming an acute angle with the thigh, the heel raised from the ground and the foot resting on the ball of the big toe, so that the gastrocnemius is stretched; and if the top of the knee be now dealt a smart blow with the palm of the hand, the tension of the gastrocnemius is suddenly increased, but no perceptible reaction ensues in healthy individuals. But if rhythmic contractions of the gastrocnemius be originated voluntarily, imitating, for instance, the movements made when an infant is dandled on the knee, it will soon be apparent that these rhythmic contractions are continued independently of the will, and require indeed a distinct effort of the will to arrest them.

The neuro-muscular apparatus engaged in the production of this movement has now been *sensitized*; and a slight tap on the top of the knee is immediately followed, quite independently of any voluntary effort, by clonic contractions of the gastrocnemius, and consequent elevations and depressions of the heel and knee. The occurrence of clonic contractions of the gastrocnemius in this posture has been observed by Dr. Buzzard and Dr. Gowers, and the latter has by the application of the graphic method proved that so long as the movement continues, between six and seven contractions of the gastrocnemius take place in a second of time, each contraction being of course followed by a distinct relaxation.

Such, then, is the ankle clonus, as it appears by cultivation in healthy subjects; but the neuro-muscular mechanism which produces it is sensitized by certain diseases to such an extent as to render any preliminary cultivation unnecessary to its production. The ankle clonus becomes developed to a high degree under the same circumstances as those in which the patellar-tendon reflex is exaggerated; the disease in which it is of the



greatest diagnostic value being sclerosis of the pyramidal tract or lateral sclerosis. Under these circumstances the reaction can be most readily obtained if the operator will grasp the heel of the patient with his left hand, the knee-joint being nearly but not quite extended, while with the right hand he produces dorsal flexion of the foot by pressing against the ball of the great toe, and thus rendering tense the tendo-Achilles. If the pressure on the latter be somewhat suddenly made, the gastrocnemius almost immediately contracts and the toe is depressed; this is followed by relaxation; and the pressure of the hand being still continued the toe is elevated, and a second contraction ensues with consequent depression of the toe; and so the contractions and relaxations are continued in rhythmic sequence so long as the tension of the tendo-Achilles is maintained. This series of rhythmic contractions constitutes the ankle clonus. Two or three weak contractions generally occur after the pressure of the hand on the ball of the toe is withdrawn, but the contractions cease instantly if passive extension of the foot is produced, so that the Achilles-tendon is completely relaxed.

If the pressure on the ball of the great toe is very gradually and steadily applied, the Achilles-tendon may be rendered tense without any contraction being caused; but, under these circumstances, a slight tap on the tendon, or even on the muscle itself, initiates the clonus, which, being once started, endures as long as the pressure is maintained. Dr. Gowers has shown that a single isolated contraction of the gastrocnemius may also be set up, the Achilles-tendon being previously rendered tense, by a gentle tap over the tibialis anticus or adjacent muscle; and he proposes to call this phenomenon the "front tap contraction." He thinks that the contraction is induced by the vibrations transmitted through the anterior muscles of the leg and the inter-muscular septum acting upon the tense fibres of the soleus and gastrocnemius. But this opinion leads us to ask whether the ankle phenomenon is like the knee phenomenon of the nature of a true spinal reflex. Dr. Gowers thinks that it is not. He found, for instance, that the interval between the tap on the stretched Achilles-tendon and the commencement of the resulting contraction varied from '025 to '04 of a second; while the corresponding interval in the case of the knee phenomenon was '10 to '11 of a second, so that the relative lengths of the two periods is as 4 to 10. We have already mentioned Dr. Gowers' observation that only  $2\frac{1}{2}$  contractions of the quadriceps muscle occur in a second in those cases in which something approaching to a knee clonus is produced; while the

contractions of the ankle clonus amount to 6 or 7 in a second. These estimates also give a relationship of about 4 to 10 between the frequency of the two contractions. From these facts, Dr. Gowers infers that ankle clonus is not a true spinal reflex like the patellar-tendon reflex; and certainly, if the various estimates of the relative lengths between the tap on the two tendons and the resulting contraction are to be relied upon, no other conclusion appears possible.

There can be no doubt that afferent fibres exist in the muscles, and just as little that passive extension of the muscle will irritate the peripheral terminations of these fibres. It appears probable that muscular action is governed by a reflex arc, the circuit of which is closed by a muscular fibre, and it is not difficult to imagine that, when the muscular fibres are rendered tense, the afferent portion of the arc is placed in a state of irritation; and that the impulses generated, on being reflected by the cord through the efferent portion to the muscular fibre, induce a condition of great molecular instability. Under these circumstances it is probable that a slight vibration passing through the muscular fibre would be sufficient to liberate its energy, and to cause a contraction. Such at least appears to be the opinion advanced by Dr. Gowers\* in the very careful paper to which so frequent allusion has been made. That the afferent fibres of the muscle are placed in a state of irritation when the clonus is induced in the healthy is shown by the fact that a considerable amount of tenderness is developed in the muscles of the calf, which lasts for a considerable time; and if passive extension develops such a molecular sensitiveness to liberation of energy in the muscular fibres under the abnormal circumstances we have just described, a similar relationship doubtless exists between muscular tension and the liberation of energy under normal conditions, which must aid the production of the rhythmical contractions of the muscles, which occur in ordinary locomotion.

One great objection to this view is that, although the interval between the tap on the tendon and the contractions of the gastrocnemius, as estimated by Dr. Gowers (.025 to .054), is short, yet it is much too long for the latter to be caused by direct action on the muscles. The vibration would be communicated to the muscular fibre almost at the time that the blow is struck on the tendon, and we have seen that the period occupied in producing latent changes in the muscles prior to contraction is .01 of a second, so that it would be impossible to account on this supposition for even the shortest estimate of the interval between the tap and the contraction. Is it possible that the contraction of the gastrocnemius is governed by a double reflex arc, the superordinate arc having its centre in the cord, and the subordinate one in peripheral intramuscular ganglia? It will, however, be better in the meantime to leave the difficulty where it stands, trusting that future researches will afford a full and satisfactory explanation of all the phenomena presented by these deep reflexes.

\*A Study of the so-called Tendon-Reflex Phenomena. By W. R. Gowers, M.D., F.R.C.P.—*Medico-Chirurg. Transactions*, vol. lxii., 1879, p. 269.



The practical point to notice is that the ankle clonus is present in a marked degree in all diseases which interrupt the continuity of the fibres of the pyramidal tract, and may even appear when conduction through these fibres is arrested for a prolonged period, even although their continuity may remain unimpaired.

§ 81. *Other Forms of Clonus.*—Although the ankle clonus is the best known and most important of the rhythmical contractions obtained by suddenly increasing the tension of tendons, yet it is by no means the only instance of this kind which may be obtained. Corresponding movements of the hand may be induced in cases of the late rigidity of hemiplegia affecting the upper extremity, by grasping the tips of the fingers and pressing the hand backwards so as to produce hyperextension at the wrist.

A similar movement may also be obtained in cases of increased tension of the muscles of the foot by producing sudden passive extension of the first phalanx of the toe, the toe being then flexed by rhythmical contractions of the abductor and flexor brevis pollicis (Gowers). Dr. Gowers also describes a lateral ankle clonus caused by contraction of the peronei, and induced by passive pressure of the foot inwards.

§ 82. *Periosteal and Fascial Reflexes.*—The best known of the muscular contractions included under this heading are the contractions of the quadriceps femoris induced by gently tapping the front of the tibia near its middle. This reaction can generally be induced when the patellar-tendon reflex is greatly exaggerated. That the quadriceps contraction is not caused by a jar communicated by the patellar-tendon is shown by the fact that it takes place when the leg is extended and reposing its whole length on the bed, and consequently when the patellar-tendon is thoroughly relaxed. The tibial tap is often followed by a contraction of the quadriceps of the opposite extremity, when the pyramidal tracts of both sides are diseased. I have also observed, under these circumstances, the tap followed by an energetic contraction of the adductors of the opposite leg.

When the upper extremity is the subject of late rigidity a slight tap with the edge of the stethoscope on the lower end of

the radius is followed by contraction of the biceps, and a similar tap on the lower end of the ulna by contraction of the triceps. That these contractions do not necessarily result from the sudden flexion or extension of the forearm caused by the blow is evinced by the fact that the reactions take place when the forearm is supported, so that both flexion and extension are prevented. When the muscles of the shoulder and forearm are implicated in the rigidity, a contraction of the pectoralis major, deltoid, and biceps may be obtained by a gentle tap on the sternal end of the clavicle; and even a crossed reaction may be obtained by a tap on the clavicle of the opposite side.

A considerable number of the muscles of the scapula and shoulder contract on tapping the spine of the scapula. In a case of advanced phthisis under my care a slight tap over the costal cartilage of the third rib on the right side is followed by contractions of the muscles of the left side of the chest, and extending not simply over the pectoral muscles, but as far as those of the upper arm and the abdominal muscles.

Contractions of the erector muscles of the spine may under certain circumstances be induced by tapping the lumbar fascia; and in a case of rigidity of the scapular muscles of one side subsequent to hemiplegia at present under my observation, the slightest tap over the upper dorsal and lower cervical vertebra, or over the muscles themselves, is followed by distinct contraction of the rhomboid muscles on the affected side, while no reaction of this kind can be obtained on the healthy side. It is unnecessary to discuss the question whether all these reactions are to be regarded as true spinal reflexes. It will suffice at present to observe that, with the exception of the contraction of the pectoral muscles in the case of phthisis, the other reactions are obtained under analogous circumstances to those in which the patellar-tendon reflex is exaggerated, and consequently often afford valuable aid in diagnosis.

§ 83. *Trepidation of Extremities—Spinal Epilepsy.*—The paroxysms of violent tremulous movements which occur in the lower extremities in certain affections of the spinal cord, and which Brown-Séquard named Spinal Epilepsy, from a fancied similarity to an epileptic convulsion, are of a compound nature.



These paroxysms occur in chronic diseases implicating the pyramidal tracts, no matter whether the latter are primarily or secondarily attacked, and consequently they appear under the same general circumstances as exaggerated patellar-tendon reflex and ankle clonus. The tremulous movements indeed appear to be caused by combined contractions induced by stimulation of some of the superficial and deep reflexes of the extremities. A painful cutaneous impression, such as is caused by pinching a portion of the skin, or even a strong pinch of the tendo-Achilles, causes reflex contractions of the muscles of the lower extremities, implicating both limbs. The primary contractions appear to predominate in the flexors, but when the anterior flexors of the leg contract dorsal flexion of the foot is produced, which in its turn stretches the Achilles-tendon and causes ankle clonus; and these actions and reactions reverberating for a time through all the muscles, the lower extremities are maintained in a state of trepidation, which may be so violent as to shake the bed on which the patient reposes. This tremor may be provoked not only by a painful impression but by the voluntary efforts of the patient to raise himself in bed, and especially by all attempts at locomotion. The most violent tremors of this kind which I ever witnessed was in the case of a patient suffering from primary lateral sclerosis. The attempts of the patient to get on the night chair were attended with tremulous movements of the lower extremities so strong as to shake his body to such an extent that he had to grasp surrounding objects firmly with both hands to prevent himself from being suddenly thrown on the floor.

If, while these tremulous movements are proceeding, the toes of one foot be grasped by the hand and brought suddenly and powerfully into plantar flexion, the muscles immediately relax and the tremors cease for a time.

#### ELEMENTARY AFFECTIONS OF THE AUTOMATIC MECHANISM OF THE MUSCLES OF EXTERNAL RELATION.

The groups of symptoms which may be included under automatic affections of the muscles of external relation cannot be distinctly separated from the reflex and voluntary affections of those muscles. There are, however, disturbances of muscular

adjustments, in which both the simple reflex actions of the spinal cord, the sensory mechanism, and the efferent or voluntary nervous mechanism are normal, and yet in which complex muscular adjustments either are effected in spite of all voluntary efforts to prevent them, or fail to be effected in spite of voluntary efforts to accomplish them. It is such movements as these which are meant to be included under the name of Automatic Kinesioneuroses of the muscles of external relation. These movements are in all probability co-ordinated in the cerebellum and basal ganglia of the cerebrum. Motor disturbances of this nature may be caused by disease of some of the nervous centres or of some of the conducting paths; but the most useful and practical classification is that which divides them into affections of the peripheral, spinal, or encephalic apparatus.

#### § 84. *Peripheral Automatic Disturbances.*

*Disorder of Labyrinthine Impressions.*—Disease of the peripheral nerves generally involves either the sensory or voluntary motor fibres, or both together, so that disorder of muscular co-ordination becomes thus obscured by the more important, or at least more prominent, disorder of the conscious muscular adjustments. It would appear, however, that the seventh pair of cranial nerves contains afferent fibres which are not subservient to the conduction of sensory impressions, and yet disease of which gives rise to phenomena of motor inco-ordination.

When the horizontal membranous semicircular canal of the internal ear is cut through in a pigeon, the bird is observed to be continually moving its head from side to side, especially during attempts at locomotion. If one of the vertical canals be cut through, the movements are up and down; and the condition is exaggerated when the canals of both sides have been operated upon. If the bird be thrown into the air, it flutters and falls down in a helpless and confused manner, and every movement which it attempts to perform is disorderly and fails of its purpose. The want of co-ordination is not due to loss of auditory sensations, since the animal can hear perfectly well, although similar phenomena may also be caused by lesion of



the auditory trunk, section of which, in the frog and mammals, produces inco-ordination of movements. Similar symptoms are also observed in man in disease of the semicircular canals or of the internal ear; an affection which will be subsequently described as Menière's disease.

§ 85. *Spinal Automatic Disturbances.*—*Ataxia* is a very characteristic kind of motor inco-ordination, which is observed in diseases of the spinal cord, and constitutes the most prominent feature of locomotor ataxia. It is characterised by inability to make combined or complicated movements with certainty and precision, and in advanced cases all movements requiring intricate and delicately-balanced muscular adjustments become impossible. The motor inco-ordination usually presents itself in the most marked manner during *station* and *locomotion*. When the patient assumes the erect posture, the muscles of the calves of the legs, those of the front of the thigh, and the erector-spinae, may be observed in a state of strong tonic contraction. Partly in consequence of these contractions, and partly in consequence of a loss of harmony in the strength of the contractions of the various groups, it is necessary for the patient, even in the early stages of the affection, to support himself by the aid of a staff, or even by the aid of one in each hand; while to prevent the body being dragged backwards by the strong contractions of the erector-spinae, it is maintained by voluntary effort flexed on the thighs.

All the movements required in walking are much exaggerated in this affection. In order to advance the foot which is about to become "passive," the pelvis is rotated vertically to an unusual extent by a voluntary contraction of the abductors of the opposite thigh; but instead of the various segments of the passive leg being now flexed upon one another, so as to admit of the usual pendulum movement of the leg, the limb is projected more or less violently forwards or forwards and outwards, the heel being the last part of the foot which leaves the ground, and when the foot is again placed on the ground the heel is brought down with a forcible thump. The phenomena differ greatly according to the degree of the ataxia. In slight cases the various segments of the passive leg may be slightly flexed on one another

during the forward movement, so that the heel may even be raised off the ground before the toe; but even in slight cases the heel is brought down to the ground with a certain degree of force. In the more severe cases the movements of the legs are exaggerated, impulsive, and jerking, often made in a wrong direction, and always with a degree of force quite disproportionate to the adjustment to be effected. The want of harmony between the degrees of strength of the various muscular contractions necessary for normal locomotion is much increased when the patient endeavours to walk in the dark, or closes his eyes. As the disease advances it becomes necessary for the patient to exercise an increased control with his eyes over the movements of his legs, so that during locomotion the eyes are kept constantly directed to the ground. During the whole of this time there is no loss of voluntary power. When the patient is lying down he can perform all the simple movements of the legs with ease, and probably with even more than normal force, since the muscular masses of the legs are not unfrequently increased in size in the earlier stages of the affection. But even in the recumbent posture the patient is unable to perform any complicated movement. If he is asked, for instance, to touch an object with the toes, the mark is generally missed. In advanced cases this uncertainty extends to the simple movements, so that on attempting even to raise the leg it is jerked hither and thither, and often in a totally different direction to the one intended.

Locomotor ataxia is caused by disease of the posterior columns of the spinal cord; and as the fibres of the posterior roots of the nerves are usually implicated, the ataxic symptoms are generally associated with various sensory disturbances, and some authors maintain that the motor inco-ordination is due to disease of the sensory apparatus. Against this view, it is urged that there frequently exists a great disproportion between the intensity of the sensory disturbance and the degree of ataxia; cases being recorded in which the ataxic symptoms were well marked, while the sensory disorders were slight or absent; while other cases manifest severe sensory disorders, probably as long as twenty years prior to the appearance of the ataxia. Other authors appear to attribute the ataxic symptoms to disease of the cerebro-spinal efferent system, which causes an abnormal strength of nervous impulses to be sent to each of the muscles engaged in effecting complicated muscular adjustment. My own opinion, however, is that the ataxic symptoms are caused by disease of the



cerebello-afferent conducting paths in the spinal cord. The undue amount of tonic contraction of the muscles of the calf, front of the thigh, and of the erector-spinae, in the early stages of the disease, would appear to indicate that at that time the irritability of the cerebello-afferent fibres of the cord is increased; while in the later stages of this affection, although there is no cerebral paralysis, yet there is cerebellar paralysis, and the consequent abolition of the tonic muscular contractions regulated through the cerebellum, overthrows the balance of the delicate muscular adjustments necessary for the maintenance of the erect posture and for locomotion; the muscles of the trunk and limbs are not maintained in that state of balanced and continuous contraction which will enable the alternate contractions regulated through the cerebrum to act efficiently and harmoniously.

*Bauch-Romberg Symptom.*—Another symptom which is closely allied to ataxia is one which, from the observers who first described it, is often called the Bauch-Romberg symptom. If a patient suffering from ataxy be made to stand with the feet close together so that they touch along the whole length of their inner margins, and if the patient be now asked to close his eyes, he immediately begins to sway from side to side; and in severe cases the patient totters, and would instantly fall unless he open his eyes or be supported. This symptom is also usually associated with diminution of the various forms of sensibility of the skin, muscles, and joints, and some, probably most, authors think that this symptom at least is due to loss of the sensory control of the lower extremities, a loss which may be partly supplied by the use of the eyes, but which immediately leads to disorderly muscular contraction when the eyes are closed. In a case at present under my care, however, the swaying movements on closing the eyes are extremely well marked, being out of proportion to the degree of ataxia, yet sensory disorders are almost entirely absent in the lower extremities. For my own part I think that these swaying movements are of the same nature as the ataxia, and that the former are caused by disease of the same kind of fibres as the latter, although it is probable that the individual fibres implicated in both instances may not be the same, since the swaying movement and ataxia are not always present in proportionate degree.

§ 86.—*Encephalic Automatic Disturbances.*

Disease or injury of the cerebellum and its connections gives rise to the most pronounced phenomena of motor inco-ordination. Flourens observed that when a small portion of the cerebellum was removed from a pigeon, the animal's gait became unsteady and disorderly; and removal of the whole organ was followed by a total loss of co-ordination. Similar experiments repeated in other animals have led to essentially the same results; and it has been found that lateral lesions and incisions produce a greater result than median incisions. Similar motor disturbances have been observed in man in tumours and other lesions of the cerebellum.

(1) *Reeling*.—The well-known gait of a drunken man is called *reeling*. It consists essentially, as Dr. Hughlings Jackson has pointed out, of swaying of the trunk from back to front and from side to side; and depends upon paresis of the tonic contractions of the muscles of the trunk, which maintain the erect posture. "The legs act erratically," says Dr. Hughlings Jackson; "but, in an early stage, they are blameless. They act erratically because they have to run after the trunk, to prop it up in its various over-inclinings."\*

(2) *Cerebellar Rigidity*.—This form of rigidity is found associated with tumour of the middle lobe of the cerebellum (Hughlings Jackson). The spinal muscles are first affected, drawing back the head and curving the spine, but after a time the legs and arms become rigid.

*Cerebellar Tetanic Seizures*.—Tumour of the middle lobe of the cerebellum also gives rise to attacks of tonic spasm similar to those of ordinary surgical tetanus. Dr. Hughlings Jackson thinks that the rigidity in cerebellar disease is due to unantagonised cerebral influx; being thus the converse of the late rigidity of hemiplegics, the latter of which, he thinks, is due to unantagonised cerebellar influx. The tetanic seizures in cerebellar diseases, Dr. Jackson thinks, are due to a discharge from the cortex of the cerebellum; just as unilateral epileptoid seizures are due to a discharge from the cortex of the brain.

(3) *Vertigo* is a sensation of swimming in the head, during

\* The British Medical Journal, February 7, 1880, p. 197.



which surrounding objects appear to oscillate before the eyes, or to rotate in a definite direction, and which is also accompanied by a sense of staggering or of rotation of the body. Vertigo appears to be the subjective correlative of loss of co-ordination between the various muscular adjustments necessary for maintaining certain attitudes in space, and of those which are active in determining the position of the body in space. Vertigo is a prominent symptom of the diseases in which the automatic mechanism for maintaining the erect posture is deranged, such as Menière's disease, and various diseases of the cerebellum. The position of the body in space is mainly determined by the association of objects seen, with the appreciation of the position of the eyes and head. Displacement of the position of the eyes, such as occurs in paralysis or spasm of one or more of the recti muscles; or of the position of the head, in such as occurs in conjugate deviation of the head and eyes, and the compulsory movements about to be described, is also accompanied by severe vertigo. This symptom frequently attends visceral disease, as dyspepsia; and it is then probably caused by vaso-motor changes in the cerebral circulation. This opinion is rendered all the more probable from the fact that vertigo is a troublesome symptom both of cerebral anæmia and congestion. Vertigo being a subjective sensation should properly have been described amongst the *æsthesioneuroses*; but its true character, as the subjective correlative of disorder of various automatic adjustments, will be best appreciated by describing it in this place.

(4) *Compulsory or Forced Movements—Co-ordinate Cramps.* In experimental injury to various parts of the medulla, pons, and crura cerebri, what are described as *forced* movements are frequently observed. It is probable that in many of these cases one or other of the peduncles of the cerebellum has been injured. One of the most common forms of these movements is that in which the animal rolls round the longitudinal axis of its own body. It generally results from section of one of the crura cerebri or unilateral section of the pons, but has also been observed after injury to the medulla oblongata and corpora quadrigemina. Another form is that in which the animal continually moves round and round in a circle, this circus movement being executed sometimes towards and

sometimes away from the injured side. Phenomena of essentially the same character, although not carried to the same extent, are observed in man as the result of disease, as, for example, what will be afterwards described as conjugate deviation of the eyes in certain cases of apoplexy. In another form of forced movement the animal rotates round the transverse axis of the body, tumbling head over heels in a series of somersaults. This variety has been observed after injury to the corpora quadrigemina and corpora striata. Nothnagel has found that after injection of chromic acid into a limited portion of the corpus striatum, which he has called the *nodus cursorius*, in rabbits, the animal runs in a straight line either backwards or forwards until stopped by an obstacle. It will be observed that some of these forced movements occur after injuries which appear limited to the cerebral hemispheres; but it must be remembered that conducting paths must exist between the cerebellum and the cortex of the cerebrum; and it is very probable that the central lesion in these cases really injures these conducting paths. It may therefore be said with a great deal of probability that in many forms of compulsory movements, in reeling, ataxia, and other symptoms of motor inco-ordination, it is the cerebello-spinal mechanism which is at fault; and that the functional disturbances are determined primarily by disorders of the tonic muscular contraction by means of which the various attitudes of the body are maintained.

Another series of motor disorders appears to depend upon disease of the cerebro-spinal system. Amongst these may be mentioned the peculiar disturbances of locomotion which occur in paralysis agitans, and the irregular movements of chorea, and various other disorderly movements. It is obvious that the pathology of these motor disturbances cannot be satisfactorily discussed at present, and must be referred to the section devoted to the special diseases.

#### 87.—*Synkinesis*.

Under this term are generally included certain involuntary movements of paralysed parts; but I shall extend the meaning of the word so as to include also certain motor anomalies which occur in muscles subject to spasm; and, in addition,



I shall include certain anomalous movements which take place in the muscles, that, in health, are associated in their actions with those primarily affected. There is certainly nothing in the derivation of the term synkinesis to forbid this extension of its meaning.

§ 88. *Associated Movements of Paralysed Parts.*—In facial paralysis of cerebral origin, the muscles of the paralysed half of the face may occasionally perform the movements necessary to changes of expression in association with those of the opposite side; although, as a rule, the contrast between the actions of the two sides is rendered all the more evident under changes of expression. In cases of hemiplegia automatic movements may occur in the paralysed arm when the patient sneezes, even when he is quite unable to move the arm by a voluntary effort; and under the influence of excitement the paralysed extremities may be strongly flexed, while the unaffected limbs remain passive. When the muscles of the paralysed side have become the subject of permanent contracture, a movement about to be voluntarily performed by the unaffected extremities may be initiated by contraction of the corresponding muscles of the opposite side. This form of associated movement is apt to occur when the hemiplegia dates from childhood. In a man at present under my care, who is slowly and partially recovering from right hemiplegia, every effort to grasp my hand strongly with his left or sound hand is accompanied by flexion of the fingers of the partially paralysed hand; and conversely, every effort to grasp strongly with the right hand is accompanied by flexion of the fingers of the healthy hand. During slighter efforts both the paralysed and healthy hand can be separately closed without any movement occurring in the other hand. In another case of hemiplegia of old date under my observation, the posterior third of the deltoid of the left side is in a state of contracture, and the upper arm is directed obliquely outwards and backwards. When the patient is at rest in the erect posture the shortest distance between the elbow and the side is five inches; but on walking, the posterior third of the deltoid immediately becomes more tense and prominent, and the elbow is projected another inch and a half outwards and backwards,

the distance between it and the side being now six and a half inches.

In all these instances, and many more of a similar kind which might be related, the localisation of the lesion is such as to interrupt conduction, either totally or partially, through the pyramidal tract, either in the course of the fibres or at their origin in the cortex of the brain; while the connection of the muscles with the spinal cord is unaffected. In this form of paralysis the reflexes often manifest increased activity, and it is also probable that cutaneous reflex influences may be conveyed from the sound to the paralysed side through commissural fibres connecting the nerve nuclei of the two sides in the spinal cord. But it is equally possible that automatic impulses from the cerebellum and basal ganglia, and even cerebral impulses from the cortex of the healthy hemisphere, may find their way to the paralysed limbs through these commissural fibres. But the whole of this subject will be better understood when we describe the relative immunity from paralysis manifested by some muscles in comparison with others, and the mechanism by which this result is brought about.

§ 89. *Relative immunity of some Muscles from Paralysis, and their relative liability to Convulsion in Cerebral Disease.* It may be laid down as a general proposition that the muscles of one lateral half of the body are regulated from the cerebral hemisphere of the opposite side, and that when the connection between the muscles of one side of the body and the cortex of the opposite hemisphere is severed paralysis of those muscles will result; or, in other words, there will be hemiplegia of the lateral half of the body opposed to the diseased hemisphere. This rule is, however, liable to many exceptions, and it is these exceptions which give the key to the interpretation of the motor anomalies which I have grouped under the name of synkinesis.

The most notable exception to this rule is, that in cases of hemiplegia while some of the muscles are liable to be completely paralysed others are only partially paralysed, while some manifest only a slight degree of feebleness. In left hemiplegia, for instance, the muscles of the extremities may be completely



paralysed; but the muscles of the trunk, especially those engaged in carrying on respiration and other automatic actions, almost entirely escape. Two reasons may be given for this variation in the degree of paralysis. In the first place the movements of respiration are well organised, they are regulated principally from the medulla oblongata, and are in large part independent of cerebral influence; while the more complicated movements of the extremities, and especially those of the hand, demand a relatively larger amount of voluntary control. The spinal nuclei of the muscles of the extremities must be therefore connected with the cortex of the brain by a much larger number of fibres than are the respiratory nuclei; and while the fibres which connect the latter and the cortex belong to the fundamental system, a large proportion of those which connect the former and the cortex belong to the accessory system, and are consequently the more vulnerable of the two. The relative immunity from voluntary paralysis of the muscles supplied by the upper division of the facial nerve, as compared with those supplied by the lower division, is also very instructive in this respect. In hemiplegia affecting the facial nerve, the muscles of the mouth and nose, which are subservient to facial expression, become completely paralysed; while the orbicular muscle of the eyelid and the occipito frontalis muscles are almost entirely unaffected. Now the movements of the muscles about the eyelids are well organised in animals and in the child at birth; the most important of these are reflex in character, such as closure of the eyelids on irritation of the conjunctiva, and they are largely independent of the will; consequently, relatively few fibres are necessary to connect their nucleus in the medulla with the cortex of the brain, and even these fibres must belong to the fundamental system. But the movements of facial expression are not well organised in animals or in the child at birth, while in the adult man they are largely under voluntary control; hence a relatively larger number of fibres must connect their nucleus in the medulla with the cortex of the brain, and a large proportion of these fibres must belong to the accessory system. It may, indeed, be laid down as a general rule that movements which are well organised in the human infant at birth, and which man possesses

in common with a large number of the lower animals, are represented in the pyramidal tract by comparatively few fibres, and that these are completely developed at birth, and therefore belong to the fundamental system. On the contrary, the movements which are acquired by man after birth, and which differentiate him from the lower animals, are represented in the pyramidal tract by a relatively large number of fibres that either do not exist at birth or exist only in a rudimentary condition, and consequently belong to the accessory system. It would appear, therefore, that both the relatively small number of the fundamental fibres of the pyramidal tract and their better organisation must render them less liable to disease than the accessory fibres, and this explains to some extent the relative immunity from paralysis of those muscles which are engaged in effecting the earlier organised movements of the body.

But the second reason for the relative immunity of certain muscles from voluntary paralysis is even more important than the first. Dr. Broadbent was the first to draw attention to the fact that the muscles which remain comparatively unaffected by paralysis in hemiplegia are those which are associated in their actions with the corresponding muscles of the opposite side, as the muscles of respiration; while the muscles which suffer most from paralysis are those which are engaged in effecting movements which are quite independent of the movements of the opposite side of the body, as the muscles of the hand. Dr. Broadbent also further conjectured that the muscles which are associated with corresponding muscles on the opposite side in their action are innervated from both cerebral hemispheres, so that severance of the connection between the spinal nuclei of these muscles and the cortex of one hemisphere still leaves the connection with the cortex of the other hemisphere intact. He has suggested that the connection with the two hemispheres is usually affected by means of spinal commissural fibres. In *Fig. 12*, for instance, *d'*, representing the spinal nuclei of the dorsal nerves of the left side, is connected with the cortex of the opposite side by fibres (5 5) which ascend in the pyramidal tract; but *d*, the spinal nuclei of the right dorsal nerves, is not only connected with the cortex of the opposite, but also with that of the same side through *c'''*, the commissural



FIG. 12.

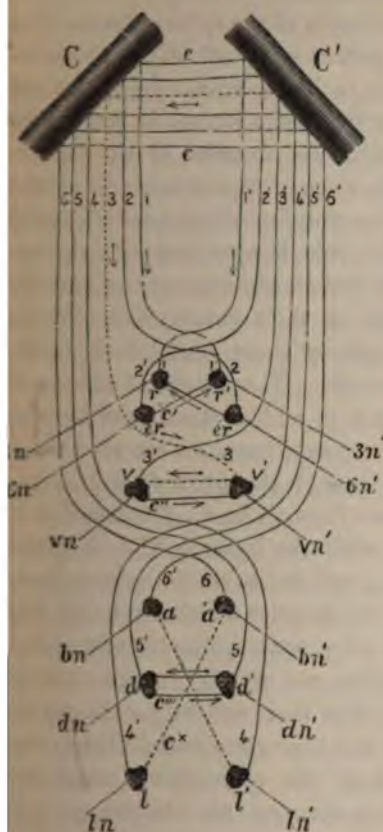


FIG. 12.—C, C', cortex of right and left cerebral hemispheres respectively; 1, 2, 3, 4, 5, 6, fibres of the pyramidal tract uniting C, the cortex of the right hemisphere, and  $r, r', v, v', a, d, l$ , the respective spinal nuclei of the internal rectus, and the external rectus muscles of the eye, the muscles of articulation and vocalisation, those of the upper extremity, the dorsal muscles, and those of the lower extremity, all of the left side;  $1', 2', 3', 4', 5', 6'$ , fibres of the pyramidal tract, connecting the cortex of the left hemisphere with  $r, r', v, a, d, l$ , the spinal nuclei of the right side corresponding to those already enumerated on the left side;  $c, c'$ , fibres of the corpus callosum uniting identical regions of the two hemispheres;  $c'$ , commissural fibres connecting the spinal nucleus of the internal rectus muscle of one eye with that of the external rectus muscle of the opposite eye;  $c''$ , those connecting the spinal nuclei of the muscles of vocalisation and articulation of the two sides;  $c'''$ , those connecting the special nuclei of the muscles of the trunk;  $c^x$ , those connecting the spinal nuclei of the posterior extremity of one side with the anterior extremity of the opposite side. The arrows indicate the direction of the conduction.

fibres which connect the two spinal nuclei. Suppose now that the fibre  $5' 5'$  is ruptured, and the nucleus  $d$  is thus severed from the cortex of the opposite hemisphere, which usually controls its function, it can still obtain impulses from the cortex of the same side through  $5 5, d',$  and  $c'''$ . When, however, the muscles of opposite sides act independently of each other, such as those of the right and left hands, commissural fibres are not established between their nerve nuclei. In the nucleus ( $a$ ) of the right upper extremity, as represented in the figure, rupture of fibre  $6'$  severs the connection with the cortex of the opposite side, and no channel is established by means of which it can obtain impulses from the cortex on the same side. Dr. Broadbent has happily called this prin-

ciple the "*bilateral association of the nerve nuclei of muscles bilaterally associated in their actions.*"

The effect which this bilateral fusion of the spinal nerve nuclei of various nerves produces in certain spasmodic affections is as striking as that produced by it in hemiplegia. In unilateral chorea, for instance, while the spasmodic action is limited to one side in the extremities and lower muscles of the face, it extends to the muscles of both sides of the trunk and of the eyelids and eyebrows, these being muscles which are habitually associated in their actions. This rule is also true with respect to other forms of convulsion, so that in *hemispasm* the muscles which are habitually associated in their actions are affected on both sides; while in *hemiplegia* these muscles are comparatively preserved from paralysis. The reason of this is so plain that it is scarcely necessary to add another word by way of explanation. Suppose that excessive impulses are sent down from the cortex of the brain through the fibres 6' and 5', the former will reach *a* and pass out through *bn* to the muscles of the arm on the opposite side; while the latter will reach *d* and pass both through *dn* and *c''*, *d'*, and *dn'* to reach the muscles of the trunk on both sides. But the muscles which are habitually associated in their actions are not always symmetrically placed on the two sides of the organism, nor are their actions always analogous. It is only necessary that their actions should concur to produce a definite result; and the commissural fibres often connect, not two nerve nuclei on the same spinal level, but nuclei at different levels, thus forming an oblique crossed connection as in (*Fig. 12, c\**).

§ 90. *Conjugate Deviation of the Eyes, and Rotation of the Head and Neck.*—The actions of the external rectus of one eye and of the internal of the other is a good example of muscles having quite opposite actions and yet concurring to produce a harmonious result. It is evident that if commissural connections exist anywhere they must exist between the nucleus of the sixth nerve of one side and the portion of the nucleus of the third nerve which presides over the action of the internal rectus of the opposite eye; and as these nuclei are placed at different levels in the pons and crura, the connection between them must be



oblique and crossed. In *Fig. 12*, let  $r$  and  $r'$  represent respectively the nucleus of the portion of the third nerves ( $3n$  and  $3n'$ ) supplied to the right and left internal recti, and  $er$  and  $er'$  be respectively the nuclei of the right and left sixth nerves ( $6n$  and  $6n'$ ); while  $c'$  represents the crossing of the commissural fibres. The external rectus of one eye and the internal of the other eye acting simultaneously rotate both eyes so as to direct the axes of vision to lateral objects. When the object is placed to the right it is manifest that the right eye is in a better position to catch the first glimpse of it, hence the external rectus which rotates the right eye outwards takes the lead in the action (Broadbent). But the internal rectus of the opposite side rotates at the same time the left eye inwards; and to effect this movement it will be a clear gain of time, as well as economy of force, if it were to receive its impulses to action through the short commissural fibres which connect the two nuclei, and not from the cortex of the cerebrum of the opposite side. When, therefore, the eyes are directed by a voluntary effort to the right, the impulse to action may be supposed to come from the cortex of the brain ( $C$ ) on the opposite side, to pass out through the fibres ( $2'$ ) of the pyramidal tract which connect the cortex with the nucleus of the sixth nerve ( $er'$ ), and then to pass on through the commissural fibres ( $c'$ ) to the part of the nucleus ( $r$ ) of the opposite third nerve concerned in the action. According to this statement, therefore, in directing the eyes laterally, say to the right, both the right external rectus and the left internal rectus receive the impulse to action from the cortex of the left hemisphere, the impulses of the nucleus of the third nerve being received through the commissural fibres which connect it with the nucleus of the sixth nerve of the opposite side. So far we have only spoken of the two recti muscles, but when these muscles are contracting so that the eyes are directed laterally, the muscles which rotate the head also become contracted in such a way that the head is turned in the same direction as the eyes, this movement being frequently observed when a man looks over his shoulder. Rotation of the head, say to the right, is produced mainly by contraction of the right inferior oblique muscle of the neck, although the left sterno-mastoid, and probably

other muscles, co-operate in the movement, and these muscles also receive their voluntary impulses to action through commissural fibres which connect their nerve nuclei with the nucleus of the sixth of the right side. According to this supposition, when a strong impulse is sent from the left cortex (C') of the brain through the fibres (2') which connect it with the nucleus (*er*) of the sixth nerve of the opposite side, these impulses will also pass through commissural fibres to the nuclei of the nerves which supply the internal rectus and sterno-cleido-mastoid muscles of the opposite side, and of the inferior oblique muscle of the neck of the same side; and the eyes and head will consequently be strongly rotated to the right, and away from the hemisphere from which the impulses originated. But this lateral deviation or conjugate deviation of the eyes, as it is called, occurs frequently in disease, and it is then associated with rotation of the head and neck to the same side as the eyes are directed. This position of the eyes and head is almost a constant accompaniment of convulsions of cerebral origin, and when the convulsions are unilateral and due to disease of the cortex of one hemisphere, the rotation always takes place towards the convulsed side and away from the seat of the lesion. Unilateral convulsions are often associated with a certain degree of hemiplegia, the convulsions being then limited to the paralysed side; and when, under these circumstances, conjugate deviation of the eyes occurs, the rotation is always *towards* the paralysed side. This, then, constitutes *spasmodic* lateral deviation of the head and eyes. But Grasset has drawn attention to the fact that this lateral deviation is often of *paralytic* origin. Let us now suppose that the fibres (2') which connect the left cortex (C') and the right nucleus of the sixth (*er*) are suddenly interrupted, the cerebral impulses to the nucleus are arrested, the external rectus of the right eye becomes paralysed, and that eye is rotated to the left. But the impulses through the commissural fibres which connect the nucleus of the right sixth, and those of the left internal rectus, and of the rotators of the head to the left must also be arrested, so that the latter muscles likewise become paralysed; hence the left eye and the head become rotated to the left, the rotation now



taking place *away* from the paralysed side and *towards* the hemisphere of the brain, in which the disease is situated. The rotation of the eyes in this direction has been facetiously described as an attempt on the part of the patient to inspect the cerebral lesion, which is the cause of the paralysis. The rotation of the eyes, head, and neck is not now due to spasm of the muscles engaged in producing the action; but to paralysis of their antagonists. This symptom is usually associated with all sudden and severe attacks of hemiplegia; it is generally absent in the slighter forms of the attack, and in all cases in which the paralysis is more or less gradual in its onset. The phenomenon is also, as a rule, a very transitory symptom of paralysis, and usually disappears in from four days to a week. The rotation of the head generally disappears first, and then the deviation of the eyes improves; but it not unfrequently happens that a temporary squint may be observed during the progress of the rotation of the eyes towards recovery.

The reason of the temporary character of the paralytic form of conjugate deviation of the eyes and rotation of the head and neck—say towards the right—appears to be that, although the nucleus of the left third (*r'*) usually receives its impulses to action through the commissural fibres which connect it with the nucleus of the right sixth nerve (*er*), and consequently from the cortex of the left hemisphere, yet channels of communication (1) still exist between the nucleus of the left third and the cortex of the right hemisphere. There is no congenital deficiency of the channels which connect the cortex of the right hemisphere and the nucleus of the third nerve of the opposite side; nor indeed of the oblique commissural fibres which connect the latter with the nucleus of the right sixth nerve; and now that the more usual channels are interrupted by disease, impulses begin to pass through the less-used channels. In a few days, then, the channel (1) between the right cortex and the nucleus of the left third nerve becomes patent, and some days later the commissural fibres (*c*) between the two nuclei become so far open as to convey impulses from the nucleus of the left third to that of the right sixth, so that the paralysis of the muscles supplied by these nerves disappears. If, however, a lesion in the pons interrupts the commissural fibres

(c) so as to prevent impulses passing from one nucleus to another, a lesion of the hemisphere will then produce a paralytic conjugate deviation of the head and eyes towards the diseased side of the brain which remains permanent (Broadbent).

§ 91. *Secondary Deviation of the Sound Eye.*—In paralysis of one of the ocular muscles, say of the external rectus of the right side, the eye is of course subject to internal squint. Now, if during recovery from this condition, when the conduction through the sixth nerve (6n) is still delayed, the eye of the sound side be closed and the patient be directed to look at an object with his right eye in such a way as to strain the external rectus muscle, this strain is accompanied by a strong voluntary effort; but owing to the diminished conductivity of the nerve only a relatively small amount of the voluntary impulses will pass to the muscle. But the impulses generated by the strong voluntary effort will pass through the commissural fibres (c') to the nucleus of the left third nerve (r') in an undiminished degree, so that the internal rectus of the left eye becomes strongly contracted. The energetic contraction of the internal rectus of the left eye induces a secondary squint in it, the extent of which is much in excess of that of the squint of the paralysed side. But although this secondary deviation is more apparent in the case of paralysis of the ocular than in paralysis of other muscles, yet essentially the same phenomenon occurs in the extremities. If the common extensor muscle of the toes is partially paralysed a voluntary effort to extend the toes is followed by flexion of them. A simple movement like flexion at the elbow joint is not caused by contraction of the flexors only, but by the predominance of their contractions over the contraction of the extensors simultaneously induced. During recovery from an attack of hemiplegia it often happens that when the patient makes an effort to flex the forearm the flexor muscles may be observed to contract, yet either no movement or movement in the opposite direction occurs; because the balance of the innervation to the antagonistic muscles is equal, or the innervation to the extensors is in excess of that to the flexors.



§ 92. *Disorders of the Associated Movements of the Extremities.*—We have seen that the movements of the limbs, and especially of the hand of one side, are largely independent of those of the other, and consequently that the spinal nuclei of the nerves which supply the limbs are not intimately connected by transverse commissural fibres. But in walking, the movement of the right leg is always associated with swinging of the right arm, and, conversely, that of the left leg with swinging of the right arm. It may be inferred, therefore, that the nuclei of the nerves of the upper ( $\alpha \alpha'$ ) and lower extremities ( $l l'$ ) are connected by oblique and crossed commissural fibres. In man the movements of the leg of one side are not very intimately associated with that of the arm of the opposite side; hence the commissural fibres, which connect their respective nerve nuclei, are represented by dotted lines ( $e^x$ ). In quadrupeds, however, the crossed association between the movements of the anterior and posterior extremities is much more intimate than in man, and consequently the oblique commissural fibres are patent in a corresponding degree.

Let us now suppose that the fibres ( $4'$  and  $6'$ ), which connect the cortex ( $C'$ ) of the left hemisphere with the spinal nuclei ( $\alpha, l$ ) of the right extremities, are ruptured. Rupture of these fibres would produce hemiplegia in man; but in the dog only a certain amount of paresis results, inasmuch as the right hind limb receives impulses through the open commissural fibres, which connect the spinal nuclei of its nerves with the nuclei of the nerves of the left anterior limb. The right anterior limb likewise becomes innervated through the commissural fibres which connect the nuclei of origin of its nerves with those of the nerves of the left posterior extremity. All the limbs of the dog therefore become innervated from one hemisphere when the other hemisphere is injured, so that, although disease of one hemisphere causes a certain amount of paresis, no true paralysis or hemiplegia results as in the case of man. This condition has often been induced by experimental lesions of one of the hemispheres in the dog, and it is always associated with conjugate deviation of the head and eyes, showing that both phenomena are induced by disease of the same mechanism. But although the dog does not manifest complete paralysis of the muscles

of the side opposite the lesion—say the right side, the lesion being in the left hemisphere—yet, on standing, a slight degree of pressure on the left side pushes the animal over to the right, the vertebral column is arched with the convexity towards the right, showing a predominance of the action of the left erector-spinae over their antagonists, and the eyes and head are rotated to the left, a position which indicates paresis of the muscles, which produces rotation of them to the right. Under these circumstances, when the dog endeavours to advance he begins to move round his tail, a movement which has been called "*mouvement de manège*," and which is the equivalent of hemiplegia in man. It is therefore probable that some of the compulsory movements described as Automatic Kinesioneuroses really belong to the Synkineses, as at present defined.

§ 93. *Disorder of the Associated Movements of Articulation.*—But when the muscles which are bilaterally associated in their action are small, and when minor nervous discharges only are requisite to throw them into action, the connection of the muscles of the two sides with one hemisphere may be brought into such habitual use that the connection with the other hemisphere, although still existing, is held practically in abeyance. The muscles which are concerned in executing the movements of articulation, for instance, are bilaterally associated; the necessary adjustments demand great delicacy of execution but no great muscular exertion; the muscles engaged in executing the most delicate of these adjustments are small; and consequently these muscles fulfil all the conditions just mentioned.

It is now a matter almost of daily observation that the muscular adjustments concerned in articulate speech are regulated from the left hemisphere; but it by no means follows that the regulation of all the functions performed by these muscles is similarly restricted. The contractions of the laryngeal muscles concerned in vocalisation, for instance, are not necessarily interfered with, because the delicate adjustments required in articulate speech are abolished; hence complete loss of the power of articulate speech is perfectly compatible with



entire absence of voluntary paralysis of any of the muscles engaged in articulation. It is not the power of producing voluntary contractions of these muscles which is lost, but the power of producing highly complex combinations of these contractions. If we suppose that  $v$  and  $v'$  are the spinal nuclei of the nerves ( $v\ n$ ,  $v\ n'$ ), which supply the muscles of articulation, the two nuclei are practically fused into one by transverse commissural fibres ( $c''$ ); and consequently impulses which start from the left cortex ( $C'$ ), and pass through the fibres ( $3'$ ) to the spinal nucleus ( $v$ ) of the right side, readily reach the left nucleus ( $v'$ ) through the commissural fibres ( $c''$ ). But, as the muscles concerned in articulation act always bilaterally and symmetrically, the channels of communication between the spinal nuclei of their nerves and the cortex of one hemisphere are brought into habitual use; while the channel of communication of these nuclei and the opposite hemisphere become partially obliterated from disuse, and probably not thoroughly developed from the first. The channels of communication between the right cortex ( $C$ ) and the nuclei  $v$  and  $v'$ , for instance, are represented by the dotted line ( $3\ 3$ ), and the commissural fibres which convey impulses from the left to the right nucleus by the dotted line ( $c''$ ), in order to indicate that these channels are only partially open. Destruction of the communication ( $3'$ ) between the left cortex ( $C$ ) and the right nucleus ( $v$ ) is followed by loss of articulate speech, a condition which is called *aphasia*.

If the lesion destroys the portion of the cortex of the left hemisphere—the posterior part of the third frontal convolution—from which the fibres of communication spring, this condition is permanent, except perhaps in young people, in whom the corresponding part of the right hemisphere becomes educated and developed for the purpose. But if the lesion involve only the channel of communication ( $3'$ ) between the left cortex and the right nucleus, the loss of speech is only temporary. The corpus callosum consists of fibres ( $c\ c$ ), which connect symmetrical parts of the two hemispheres; and the portion of it which connects the third frontal convolution of the two sides is represented in Fig. 12 by the dotted line, to show that although the connection exists it is partially closed through disuse. When, however, the communication through ( $3'$ ) is interrupted,

impulses generated in the third left frontal convolution make their way through the fibres of the corpus callosum to the corresponding part of the right hemisphere, and after a time through the dotted line (3), which connects the latter with the left nucleus, and, after another interval, through the partially open commissural fibres which connect the left (*v*) with the right nucleus (*v'*), so that the power of speech is gradually re-acquired. A lesion, however, which destroys both the channel of communication (3') between the third left frontal convolution and the spinal nuclei, and the fibres of the corpus callosum (*c c* dotted line), connecting the right and left third frontal convolutions, will influence speech as powerfully and permanently as disease of the grey substance of the third left frontal convolution itself. Such a lesion effectually cuts off the third left frontal convolution, in which the higher mechanism which regulates the muscular adjustments concerned in articulation is organised from the spinal nuclei; and the only means by which speech can be then restored is the organisation of a new mechanism in the corresponding part of the right hemisphere, a method which must always be slow, and which can only take place, at least to any considerable extent, in the plastic tissues of young people.

For this exposition of the phenomena, which I have grouped together under the term *Synkineses*, I am myself responsible; but the whole of what I have said is inspired by the succession of able contributions on the subject from the pen of Dr. Broadbent. To Dr. Broadbent, indeed, we owe the enunciation of the principle of the bilateral association of the nerve nuclei of the muscles bilaterally associated in their action, which affords the key to the interpretation of the phenomena, and the subject has been worked out by him with such fulness of detail and such consummate skill that little is left to others in this field but to copy his work.

(II).—MOTOR AFFECTIONS OF INTERNAL ORGANS (VISCERAL KINESIONEUROSES).

§ 94. The motor affections of internal organs, exclusive of the disturbances of the blood-vessels, present many peculiarities in comparison with those of the organs of external relation



These peculiarities depend in great part upon the fact that the muscular apparatus of the internal organs is formed of unstriated muscular tissue, which differs from the striated muscle in its mode of contraction, and in several other respects. An unstriated muscular fibre does not respond to mechanical and electrical stimuli by a prompt contraction of short duration; but the contraction is preceded by a long latent period and lasts a considerable time, after which relaxation slowly takes place. The slow and protracted nature of the contraction of unstriated muscular fibres renders it impossible for tremor and fibrillary contractions to appear as symptoms in affections of those muscles.

Another important peculiarity of the contractions of unstriated muscles is their rhythmic and automatic character. The peristalsis of the intestines and ureters is rhythmic, and probably in great part automatic. It is very probable that these movements depend in great measure upon the presence of local ganglia in the walls of the organs, and such ganglia have been anatomically demonstrated in the heart, stomach, intestines, uterus, and other organs. Co-ordinating arrangements appear to be present between these ganglia, which regulate the contractions of the various segments of the viscus, so that they contract in orderly sequence.

The functions of these intramural ganglia are regulated by means of accelerating and retarding nerve-fibres from the cerebro-spinal system. Inhibitory nerve-fibres pass to the heart along the vagus, and to the intestines in the splanchnic nerves; while the accelerating fibres to the heart pass along the lowest cervical and uppermost thoracic ganglia of the sympathetic, and to the intestines from the sympathetic plexuses of the abdomen.

It must also be noticed that acceleration or inhibition of the visceral movements may be produced by irritation of certain centres in the cord and brain. Centres for the movements of the bladder and rectum, and for erection and ejaculation, are found in the lumbar portion of the cord, and for the movements of the blood-vessels in the entire length of the cord and in the medulla oblongata. The activity of these centres is evoked by means of peripheral irritation, and consequently their functions may be regarded as being in great part of

reflex nature. These centres of innervation are also connected by means of exciting and inhibitory fibres with the cortex of the cerebrum, so that the functions of the bladder, rectum, and other organs are brought to some extent under voluntary control.

From what has just been said, it is manifest that the conceptions of spasm and paralysis, which are applicable to the conditions of excess or diminution of the activity of the striated muscles, are not applicable to the corresponding conditions of the unstriated muscles. In the normal condition the muscular fibres of the internal organs are maintained in a state of rhythmic contraction. A due degree of this contracted condition may be regarded as the normal *tonus* of the organ. The condition of motor excess, or of increased *tonus*, may be called *hypertony*; while that of muscular weakness or relaxation constitutes *Atony* (Eulenburg). This change in the *tonicity* may be of direct or of reflex origin.

But pathological anomalies in the territory of the inhibitory fibres or of the inhibitory centres may lead to corresponding motor disturbances of the viscera. Irritation of the inhibitory fibres and centres will give rise to paresis or complete paralysis of the corresponding muscles, while inactivity of these fibres and centres will give rise to spasm or tetanus of the muscles. Disturbances of motor co-ordination of the visceral muscles, as, for instance, of erection, ejaculation, and the mechanism of urination and defæcation, may occur, analogous to the inco-ordination of the movements of the muscles of external relation observed in locomotor ataxy and other diseases.

From what has been said it is evident that the visceral motor disturbances must be more complicated than those of the voluntary muscles; and it will be useful, before proceeding further, to illustrate the nervous mechanism which regulates the movements of the internal organs by giving a somewhat detailed analysis of the innervation of one of them. The mechanism which regulates the movements of the heart is better known than that of other internal organs; and the description of this mechanism will, therefore, best serve to illustrate the general arrangements of visceral innervation.



§ 95. *Innervation of the Heart.*

The nervous centres which preside over the movements of the heart consist of the intracardiac ganglia, centres in the medulla oblongata, and centres in the cortex of the brain. The position of these centres may be illustrated by the diagram (*Fig. 13*) already employed to illustrate encephalo-spinal action; but *s* now represents a cell of a cardiac intramural ganglion, and *c* and *c'* cells of the centre in the medulla oblongata; while the centres in the cortex of the brain are unrepresented. These centres are connected with each other and with the periphery in various ways. First, afferent (*a*) and efferent (*e*) fibres connect the intramural ganglion

FIG. 13.

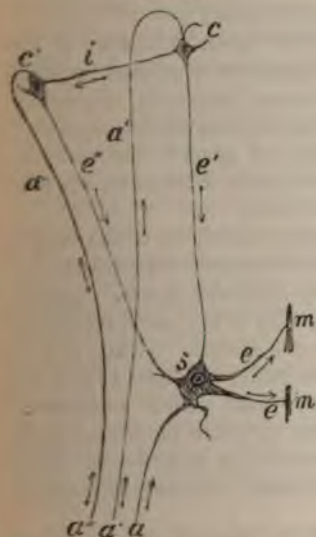


Fig. 13. *Schema of the Action of the Cardiac Nervous Mechanism.*—*s*, cell of intramural ganglia of the heart; *c* and *c'*, ganglia cells of the medulla oblongata, the former being inhibitory, the latter motor; *a*, afferent fibre to intramural ganglion cell; *a'*, afferent inhibitory or excitoinhibitory fibre; *a''*, afferent-motor or excitomotor fibre; *e'*, efferoinhibitory fibre; *e''*, efferomotor fibre; *s, s, s*, efferent fibres of the reflex arc of the intramural ganglia. The fibres connecting the ganglia cells of the medulla oblongata with the cortex of the cerebrum are not represented; the arrows indicate the direction of the conduction.

cell (*s*) with the periphery, constituting a simple reflex mechanism. Second, intercentral fibres (*e''*) connecting the cell (*c'*) in the medulla and the intramural ganglion cell (*s*), and conveying impulses which increase the activity of the heart, and hence are called *accelerating fibres*. Third, intercentral fibres (*e'*) between a centre (*c*) in the medulla and the intramural ganglion (*s*), carrying impulses which arrest the action of the heart, and are therefore called cardio-inhibitory fibres. Fourth, afferent fibres (*a''*), connecting some portion of the periphery and the motor centre in the medulla, and stimulation of which excites the centre to increased action. These fibres are, from their action, called *excito-motor*, although *affero-accelerating* fibres would best characterise their function. Fifth, afferent fibres (*a'*), which connect some portion of the periphery with the cardio-inhibitory centre in the medulla, and irritation of which renders the action of the heart slower. These fibres are called *excito-inhibitory*; but *affero-inhibitory* would best characterise their functions. Sixth and seventh, fibres (not represented in Fig. 13) connecting the cortex of the opposite hemisphere of the brain with the cardio-motor and cardio-inhibitory centres in the medulla. These fibres may respectively be called *centrifugo-*

*motor and centrifugo-inhibitory.* It will be seen, therefore, how exceedingly complicated the cardiac nervous mechanism is, and in what a variety of ways this mechanism may be deranged.

1. *Disturbances of the Simple Reflex Cardiac Mechanism.*—The heart may be thrown into action, as Bernard has shown, by stimulating the endocardium, a fact which appears to show that afferent fibres terminate in this membrane, which connect it with the intramural ganglia, and through efferent fibres with the muscle of the heart. Poisons which act on the muscular substance of the heart, as the salts of potassium, lactic acid and its salts, and various other agents, of course destroy this mechanism, but only in an indirect manner, by rendering the muscle incapable of responding to a nervous stimulus. Such poisons arrest the action of the heart in *diastole*.

2. *Disturbances of the Cardio-inhibitory Mechanism:*—

(i.) *Affections of the inhibitory fibres of the Vagus.*—The beat of the heart may be checked or entirely stopped in diastole by efferent impulses through the vagus nerve. The stimulus usually employed to generate these impulses is the interrupted current; but the same effect is produced, though less readily, by mechanical and chemical stimuli. Czermak, by pressing his vagus against a small osseous tumour in his neck, was able to arrest temporarily the beating of his own heart.

Atropia and curara paralyse the cardio-inhibitory fibres of the vagus; so that after its use the action of the heart becomes accelerated, and stimulation of the vagus produces no cardio-inhibitory action. Calabar bean, on the other hand, increases the irritability of the cardio-inhibitory fibres, so that its action is antagonistic to that of atropia. Nicotin at first slows the action of the heart even to a standstill; but after a time the beats recover their usual rhythm and may even become accelerated. Hence, it is inferred that this drug paralyzes the cardio-inhibitory fibres of the vagus, but that the paralysis is preceded by irritation of them. Muscarin at once produces a standstill of the heart, which may be removed by atropia.

(ii.) *Affections of the inhibitory centre in the Medulla.*—In acute and chronic diseases of the base of the brain the rhythm of the heart is often greatly changed, and this is probably due to irritation or paralysis of the cardio-inhibitory centre in the medulla. In tubercular meningitis, for instance, the pulse, which is at first very slow, becomes exceedingly quick towards the terminal period of the disease, phenomena which are best explained by supposing a primary irritation and secondary paralysis of the cardio-inhibitory centre in the medulla. Direct stimulation of the centre in the medulla oblongata produces inhibition of the heart.

(iii.) *Affection of the Reflex Inhibitory Mechanism.*—It would appear that powerful stimulation of any part of the body will produce reflex inhibition of the heart. Crushing of a frog's foot will, for instance, stop the action of the heart; and in man, fainting from severe pain appears to be caused by an inhibitory action on the heart. But it is probable that in the latter



instance the afferent impulses are first conveyed to the cortex of the brain and reflected thence to the inhibitory centre in the medulla, so that it can scarcely be called a true reflex action. Injury of the intestines, however, appears to exercise a more powerful effect on the action of the heart than that of any other part of the body. If the abdomen of a frog be laid bare and the intestines be sharply struck, the heart will stand still in diastole just as occurs in powerful stimulation of the vagus; and cardiac inhibition is also produced if the mesenteric nerves or their connection with the sympathetic system be stimulated with the interrupted current. When, however, both vagi are divided, or the medulla oblongata is destroyed, prior to the stimulation of the intestines or mesenteric nerves, the cardio-inhibitory action fails to be produced.

3. *Accelerator Nerves*.—After division of the vagus the heart's beat may be quickened by direct stimulation of the spinal cord. Certain fibres pass from the cervical spinal cord along the nerve accompanying the vertebral artery, and reach the heart through the last cervical and first thoracic ganglia, and stimulation of these by means of the interrupted current causes a quickening of the heart's beat; hence they have been called the accelerator nerves of the heart. Irritation of these nerves causes a quickening of the activity of the heart, while diminution of their conduction causes a slowing of the heart's beat; but abolition of their conduction does not arrest the action of the heart, because the activity of the intra-cardiac ganglia remains unaffected.

4. *Disorders of the Rhythm of the Heart*.—It is evident that a very slight derangement of the exquisitely delicate and complex nervous mechanism by which the activity of the heart is regulated must suffice to produce grave disorder in the regularity of its rhythmic contractions. Irregularity in the pulsations of the heart and other rhythmic anomalies are grouped under the term *arythmia cordis*; and this condition may be caused by a slight loss of balance between the irritability of the various centres and conducting paths of which the nervous mechanism of the heart consists.

### § 96. *Innervation of the other Viscera.*

It would occupy too much space to enter upon a similar analysis of the motor affections of the other viscera, as the various segments of the intestines, the bladder, the urinary passages, and the uterus with its appendages. Complicated affections occur in these organs, both from lesion of the intramural ganglia and of the intramuscular nerve terminations, as well as from direct or indirect irritation of the accelerator and inhibitory nerves; giving rise to spasm or paralysis, or to hypertony or atony, or to affections of co-ordination. Ischuria or constipation may, for example, result from severe cerebral or spinal disease in consequence of diminution or abolition of conduction through those cerebral paths through which motor impulses are conveyed to the detrusor vesicæ and the muscles of the rectum. The same phenomena may be produced through tonic stimulation of the sphincters of the urethra and

rectum from irritation of the reflex centres in the lumbar portion of the spinal cord ; while the converse condition of depression gives rise to incontinence of urine and involuntary passage of the stools. Erections may occur as paralytic phenomena after severe traumatic injury of the spinal cord, or as a symptom of irritation, either direct or reflex, of the genito-spinal centre in the lumbar portion of the cord. Similar motor phenomena may also occur in the intestines, uterus, and other organs.

(III).—VASO-MOTOR DISTURBANCES (VASCULAR KINESIONEUROSES, OR ANGIONEUROSES).

The calibre of the blood-vessels throughout the body appears to be regulated by means of a nervous mechanism similar in all essential respects to that which regulates the movements of the hollow viscera. The middle coat of all arteries contains circularly disposed smooth fibres, which become relatively more abundant in the smaller arteries, and contraction of which diminishes the calibre of the vessels. Nerve-fibres belonging to the sympathetic system are freely distributed to the blood-vessels, and it is supposed that numerous excito-motor ganglionic elements exist in the plexus which surrounds the vessels, forming local vaso-motor mechanisms. These local ganglia, along with the fibres passing from them to terminate in the muscular fibres, form a simple co-ordinating vascular mechanism, or, in the language of Meynert, a projection system of the third order. These local ganglia are supposed to be connected with vaso-motor centres in the spinal cord and medulla oblongata by two kinds of fibres, but the conducting paths in the cerebro-spinal and sympathetic nerves are not yet clearly made out in all cases. Irritation of the one kind of fibres excites the local ganglia to increased activity, and consequently induces contraction of the muscular coat ; hence they are called *vaso-constrictor* fibres ; while irritation of the second kind arrests the action of the ganglia, and consequently paralyses the muscular coat and dilates the vessels, and hence they are called *vaso-dilator* fibres. The vaso-motor centres in the cord and medulla oblongata, along with the fibres which connect them with the local ganglia, form a compound co-ordinating vascular mechanism, the fibres, in the language of Meynert, forming a projection system of the second order. During health the action of this nervous mechanism is so balanced that the vessels



are maintained in a medium but variable degree of contraction, which constitutes the vascular *tonus*. The arterial *tonus* throughout the body is maintained and regulated by means of a general vaso-motor centre situated in the upper part of the medulla oblongata. From this centre vaso-motor fibres pass to the various vascular areas of the body, passing out partly through the *rami-communicantes* to the sympathetic system, and partly descending in the antero-lateral columns of the cord, to pass out with the anterior roots of the spinal nerves and to be distributed to the periphery through the mixed spinal nerves. It has been proved that afferent impulses from various parts of the body may exalt or depress the arterial tone by constricting or dilating either the whole vascular system or particular vascular areas. It is manifest that a mutual antagonism must exist between the local and general effect on the circulation, by constriction or dilatation of a particular vascular area. If a particular vessel is constricted, the area to which it is distributed is less freely supplied with blood, the tissues become blanched, and the temperature falls; but provided the condition of the heart continues the same, there will be an increased flow of blood through the other arteries of the body, and the general arterial pressure will be augmented; while the converse of this holds good when an artery is dilated.

The local and general effect produced may be illustrated by stimulating with the interrupted current the central end of the divided *depressor* nerve, which is a branch of the vagus running alongside the carotid artery and sympathetic nerve, while the arterial pressure in the carotid artery is being registered. When the central end of the nerve is stimulated a marked fall of the pressure in the carotid artery is observed; but when the splanchnic nerves are previously divided the fall is very slight, showing that the greater part of the effect had been produced by dilatation of the intestinal arteries caused by irritation of the depressor nerve. The afferent impulses reaching the vaso-motor centre through the depressor nerve either depress or inhibit a portion of that centre, or irritate that portion of it which is connected with the local ganglia by vaso-dilator fibres. Irritation of the central end of the *pressor* fibres, which are found in the sympathetic nerve, raises the general arterial

pressure by contracting the intestinal arteries. Stimulation of almost any afferent nerve affects the blood pressure, even when the heart's beat remains unchanged. A medium degree of irritation of the peripheral nerves, especially the cutaneous sensory nerves, causes an increase, while strong irritation causes great diminution of the arterial tonus. It is very probable that the vaso-motor centre must be regarded as a reflex and not as an automatic centre; and it is probable that the afferent fibres of the reflex arc pass upwards in the lateral columns of the cord; at least the experiments of Miescher and Nawroski appear to prove that the afferent fibres of the reflex arc for the posterior extremities in the rabbit occupy this position in the lumbar portion of the spinal cord. But, although the centre in the medulla oblongata is the general vaso-motor centre for all the arteries of the body, subordinate centres appear to exist throughout the length of the spinal cord; at least the experiments of Goltz has proved that such centres exist in the lumbar portion of the cord for the lower extremities.

Recent experiments have shown that the vaso-motor centre in the medulla is connected with certain definite regions in the cortex of the brain; these connecting paths constituting a vascular projection system of the first order. Budge found that irritation of the crus-cerebri induced arterial contraction, and similar observations have been made in various pathological conditions. Eulenburg and Landois have proved that definite regions of the cortex of the brain in dogs, corresponding to the central convolutions in man, constitute vaso-motor centres for the extremities of the opposite side of the body. These vaso-motor centres are situated in the vicinity of the voluntary motor centres of the cortex, and destruction of them with the actual cautery leads to a more or less enduring increase of temperature in the extremities of the opposite side. These centres, then, when acting upon the inferior centres, constitute double compound vaso-motor centres, and their existence explains the frequency with which vaso-motor disturbances are associated with the most various cerebral diseases.

§ 97. *Varieties of Vascular Motor Disturbances.*—Vascular motor disturbances may be subdivided into those which are



caused by excess, and diminution or abolition of motor innervation; the former giving rise to contraction of the arterial walls or to hyperkinesis of the vessels; while the latter causes dilatation of the vascular walls, or akinesis of the vessels. The first condition may also be called *Angiospasm*, and the second *Angioparesis* and *Angioparalysis*. From what has already been said with respect to the existence of both vaso-constrictor and vaso-dilator nerve-fibres, it is evident that contraction and dilatation of the vessels may arise from very different conditions; and that the latter condition is not always to be regarded as a truly paralytic symptom. The affections of the vessels may arise from lesions either in the territory of the peripheral, spinal, or cerebral portions of the nervous system.

(1) *Peripheral Angioneuroses*.—Lesions of the peripheral nervous system and its connected ganglia give rise to both spasmodic and paralytic affections of the vessels. The disorders of innervation may be of direct or reflex origin.

The reflex disorders may be produced by lesion of the afferent fibres which pass from the vessels to the intramural ganglia in the vascular walls, or of the afferent branches from other parts of the organ to one or more of the ganglia situated higher up. Both spasm and paralysis of the vessels may be produced in a reflex manner, although it is not known under what conditions the one or the other state is caused; and there can be no doubt that these conditions are frequently active in producing local nutritive changes in organs, or in aggravating affections already existing. After injury of the peripheral nerves, especially after complete division of the large nerve-trunks of the extremities, paralysis and anaesthesia are always associated with redness and increased temperature of the affected extremities, which no doubt depend upon paralysis of the vaso-constrictors, and not upon irritation of the vaso-dilators. With degeneration of the peripheral portion of the injured nerve, trophic changes occur in the affected extremity, the circulation becomes less active, the material exchanges are diminished, and less local heat is generated, while owing to the dilatation of the superficial vessels more heat is radiated; hence the temperature of the limb falls below the healthy standard.

(2) *Spinal Angioneuroses*.—Lesions of the vaso-motor centres in the cord and medulla oblongata, and of the associated centrifugal sympathetic and cerebro-spinal conducting paths, either increase or diminish the tonic innervation, which in health is

constantly passing to the vessels; hence the resulting vascular affections may be regarded as *hypertony* and *atony*, and not as true spasm and paralysis.

The vascular disturbance may be produced, either directly or in a reflex manner, from different parts of the periphery. The vascular *tonus* may in this reflex manner be either increased or diminished for the whole or for a part only of the vascular system. Hypertony or atony of the blood-vessels may, therefore, result not only from lesions of the spinal cord and medulla oblongata, the nerve roots, rami-communicantes, sympathetic system and its peripheral branches, and the cerebro-spinal nerves; but also from affections of the skin or of the parenchymatous viscera. Whether the vascular affection is diffused over the whole vascular system, or limited to larger or smaller vascular territories, depends upon the position of the primary lesion as well as upon the duration and intensity of the reflex irritation. Even the quality of the vascular affection depends upon the degree and duration of the primary irritation. A feeble local irritation of the skin gives rise to momentary contraction of the vessels in the vicinity, with local diminution of temperature, soon followed by local vascular dilatation with increased temperature. Strong cutaneous irritation acting on a large surface produces a considerable diminution of temperature, not simply of the surface, but also of the internal organs as measured by the temperature in the rectum. This, no doubt, depends upon contraction of the vessels caused by reflex irritation of the vaso-motor centres in the spinal cord and medulla oblongata.

Injuries and diseases of the spinal cord which cause paraplegia are not unfrequently associated with primary increase of the temperature of the paralysed parts, followed by a diminution of temperature when degenerative changes occur in the peripheral nerves below the seat of the lesion and in the muscles supplied by them. Injuries of the cord which give rise to spinal hemiplegia are often accompanied by a primary increase of temperature of the paralysed side. This primary increase is especially well marked in injuries of the cervical portion of the cord near the medulla oblongata, but even this increase is generally followed by a corresponding diminution of temperature before death. In some cases, however, instead of a fall of temperature before death, it continues to rise after the injury, and may even increase considerably after death. This remarkable rise of temperature probably depends upon sudden paralysis of the whole vaso-motor system, or upon interference with the action of a heat-regulating centre in the medulla oblongata.

(3) *Cerebral Angioneuroses*.—Cerebral lesions of the cortical vaso-motor centres and of the conducting paths which connect them with the vaso-motor centre in the medulla give rise to various vascular disturbances. The pallor of fear and the blush



of shame are no doubt produced by a functional affection of the cortex acting in a reflex manner on the vaso-motor centre in the medulla.

It is also probable that many cases of unilateral hæmidrosis and ephidrosis, as well as certain vascular affections in hemicrania, Graves' disease, epilepsy, hysteria, and mental diseases, must be regarded as direct or reflex psychomotor angioneuroses.

Paralysis of cerebral origin, as in hemiplegia resulting from an apopleptic attack, is generally associated with a slight elevation of temperature of the paralysed parts, which is almost never beyond  $1^{\circ}$  C. The normal temperature is very rarely maintained, and a fall of temperature is still rarer. In long-standing cases of hemiplegia the temperature falls to the normal standard or may even sink below it. In fatal cases the temperature of the two sides, as a rule, become equal before death, but at times the temperature of the paralysed side cools sooner than the other after death. In old-standing cases the pulse on the paralysed is often smaller and more compressible than on the healthy side; and the paralysed hand and foot is whiter and colder than the corresponding parts of the other side. In some cases of cerebral paralysis the increase of temperature on the paralysed side persists for a relatively long time, and in them it is probable that this increase depends upon paralysis of the vaso-motor cortical centres, or an interruption of the centrifugal conducting paths between these and the centre in the medulla.

§ 98. *Cutaneous Angioneuroses*.—Diffused pallor and redness of the skin—the former caused by increase and the latter by diminution of the normal tonic innervation of the smaller superficial arteries—may be observed both in normal and abnormal conditions of the nervous system. Examples of diffused cutaneous pallor and redness may be observed under the action of various emotions, as fear and shame; while sudden pallor is associated with fainting, and alternating conditions of pallor and redness may be observed in various neuroses, as hysteria and epilepsy.

Some parts of the body may present vascular dilatation with increased temperature and secretion, while other portions present the opposite conditions of vascular contraction, diminished temperature and secretion. In epileptic and hysterical attacks the countenance may be flushed and covered with sweat, while the extremities are blanched and cold. These phenomena may at times be limited to one half of the body, as in many cases of epileptic aura, and are not unfrequently associated with corresponding changes and fluctuations in the pulsation of the larger superficial arteries. In these cases the reddened portions may become

quickly blanched, and conversely the pallid parts may become quickly reddened. Blushing is one of the most interesting vaso-motor phenomena, and a similar vascular condition may be closely simulated by the inhalation of nitrite of amyl. Blushing is, as Darwin remarks, of all expressions the most human. The lower animals do not blush; neither does the human infant. Idiots rarely blush, but the insane are sometimes particularly liable to blushing (Crichton-Browne). Women and children blush more than men; and the muscles of the face become dilated from the emotion of shame in almost all the races of men, though in the very dark races no distinct change of colour can be perceived (Darwin). The face, ears, and neck are the parts which redden in most cases, but in some sensitive people the redness extends to the upper part of the chest; and Diffenbach mentions the case of a lady in whom, during exposure for surgical examination, the blush extended over the nates. Most persons when blushing deeply manifest considerable mental confusion, a fact which appears to indicate that the cerebral circulation is simultaneously affected. Pallor is sometimes caused under conditions which would usually induce a blush. "A young lady told me," says Mr. Darwin, "that in a large and crowded party she caught her hair so firmly on the button of a passing servant, that it took some time before she could be extricated; from her sensations she had imagined that she had blushed crimson, but was assured by a friend that she had turned extremely pale."\* Converse facts have also been observed, in which the face has become flushed under conditions, such as sudden fright, which usually induce pallor (Eulenburg). The tendency to blushing is inherited, and Darwin, on the authority of Burgess, mentions the case of a family, consisting of a father, mother, and ten children, all of whom, without exception, were prone to blush to a most painful degree.

Various vaso-motor anomalies occur in fevers. The initial rigor of various acute diseases and the subsequent warm and moist surface, especially in intermittent fever, appear to be caused by a primary contraction and secondary dilatation of the cutaneous arteries.

*Local Anæmia and Hyperæmia of the Skin* may be caused by a direct or reflex local irritation of the surface, such as atmospheric influences, local application of cold, and various mechanical, chemical, and electrical irritants. Besides many cases of circumscribed vaso-motor aura in epilepsy as those described by Nothnagel† on the extremities, the circumscribed patches occurring on the hands and forearms of washerwomen may be mentioned, in which there is sudden pallor and coldness of the affected portion of skin, along with diminution of sensi-

\* Expression of the Emotions in Man and Animals, by Charles Darwin, M.A., F.R.S. 1872.

† "Deutsches Archiv. f. Clin. Med.," ii. 2.



bility. These patches may at times be observed in the region of distribution of a single nerve as the median, and are not unfrequently associated with trophic affections, as roseola and urticaria (Eulenburg).

What has been described under the name of *Cerebral Maculæ* (*Tâches Cerebrales* of Trousseau) consist of scattered red blotches and mottlings on the chest or abdomen of epileptics, and those suffering from Graves' disease and other neuroses.

When the affected portion of skin is rubbed, or in strongly-marked cases, is merely touched by the finger, the surface soon becomes suffused with bright red marks, which spread to some distance around the point touched, and persist for several minutes. Blushing may also occur in disseminated patches, as in the case of a mother and daughter described by Sir James Paget, in whom "a big splash of red appeared first on one cheek, and then other splashes variously scattered over the face and neck."\*

§ 99. *Visceral Angioneuroses*.—The vaso-motor nerves of the thoracic and abdominal vessels are in great part found in the plexuses of the sympathetic. The vaso-motor nerves of the thoracic viscera are derived from the inferior cervical and superior thoracic ganglia; and from the spinal cord by communicating branches between the third and seventh dorsal vertebræ. The vaso-motor nerves of the abdominal viscera, on the other hand, exist chiefly in the splanchnic nerves, but the stomach appears to be supplied with some fibres from the vagus. The splanchnic nerves are three in number—the greater, the lesser, and the smallest—and all of them arise from the thoracic ganglia of the sympathetic. The first takes its origin from the fifth to the tenth ganglia and the second from the tenth to the eleventh, and those appear to supply chiefly the stomach, liver, spleen, pancreas, and intestines. The third arises from the twelfth thoracic ganglion, and it, along with some fibres from the lesser nerve, disappears in the plexus passing to the kidneys. Section of the splanchnic nerves causes, according to V. Bezold, a great diminution of the arterial pressure caused by the great dilatation of the vessels and consequent engorgement of the abdominal viscera.

\* See Darwin's *Expression of the Emotions*, p. 312, *et seq.* 1872.

Irritation of the distal end, on the other hand, causes contraction of these vessels, and elevation of blood pressure generally. According to the experiments of Rossbach and Quellhorst\* it must be assumed that a part of the vaso-motor nerves of the abdominal viscera pass in the vagus. These authors have shown that irritation of the peripheral end of the divided vagus causes considerable increase of the arterial pressure in the abdominal vessels; even after paralysis of the intracardial terminations of the vagus has been produced by atropine.

Brown-Séquard† has shown that experimental injury of the lumbar portion of the spinal cord in guinea-pigs sometimes causes congestion of and occasionally even extravasation of blood in the suprarenal capsules. In a case of acute partial myelitis, observed by Bouchard and Béhier, besides the usual phenomena, fresh hæmorrhagic foci were found in the substance of the suprarenal capsules. Schiff, Brown-Séquard, and other experimentalists found after destruction of the pons and basal ganglia the frequent occurrence of hyperæmia and ecchymoses in the lungs, pleuræ, kidneys, and in the mucous membrane of the stomach and bowels. Crushing or section of one half of the pons causes, according to Brown-Séquard, hæmorrhages into the lung of the opposite side. The paths through which these influences traverse are not the vagi, but the sympathetic system and its spinal communications. Brown-Séquard also found hæmorrhage in the stomach after destruction of the pons at the level of the peduncles of the cerebellum. Eulenburg found intestinal hæmorrhage after burning of the upper surface of the posterior division of the cortex of the brain in a dog, although he regards the connection between the two as being somewhat doubtful. Congestion and extravasations of blood in the internal organs are not uncommon in association with hemiplegia; either in consequence of extravasation of blood or from softening. Charcot mentions a case of left-sided apoplectic hemiplegia from extravasation of blood into the right corpus striatum, in which ecchymoses were found in the pleuræ, endocardium, and in the mucous membrane of the stomach; while the Galea aponeurotica of the paralysed side assumed a wine-red colour, and showed several ecchymoses.

Various menstrual disorders, both by way of excess or diminution of discharge, which are so frequently associated with emotional disturbances, are no doubt the result of functional disorders of the vaso-motor nerves in different parts of their course in connection with general nervous

\* Centralblatt, 1876, No. 42.

† Experimental Researches applied to Physiology and Pathology, by Brown-Séquard. New York, 1853. Page 13.



affections, such as hysteria. It is also probable that many of the vicarious hæmorrhages of the stomach, intestines, lungs, and other organs depend upon disorder of the vaso-motor innervation.

§ 100. *Secretion of Urine*.—Certain changes, both quantitative and qualitative, in the condition of the urine, such as abnormal increase or diminution of its quantity, the presence of abnormal constituents, as albumen, must also be often attributed to affections of the vaso-motor nerves of the kidneys.

Bernard places the vaso-motor centre of the kidneys in the upper part of the floor of the fourth ventricle, and he has found that injury of this part causes polyuria and albuminuria, while injury of the lower part of the floor of the ventricle causes temporary glycosuria. Various derangements of the urinary secretion are not uncommonly observed after cerebral hæmorrhage, or during the growth of cerebral tumours, and these anomalies are peculiarly liable to occur in hæmorrhage of the pons. The occurrence of diabetes mellitus, and insipidus, in consequence of lesion of the nervous system, is of peculiar interest. The primary lesion in these cases may be of cerebral, spinal, or peripheral origin.

§ 101. *Glycosuria*.—Bernard first observed that injury of a circumscribed portion of the fourth ventricle is followed by the presence of sugar in the urine, and Schiff attributed the result to consecutive paralysis of the vaso-motor centre.

Recent researches have shown that irritation of a much wider area may give rise to the same phenomena. Eckhard has shown that destruction of the *vermis cerebelli* in rabbits gives rise to the presence of sugar in the urine, which is not attended by any alteration in the blood pressure, and consequently is not likely to be caused by disturbances in the circulation. Irritation of the vermis only gives rise to hydræmia when the vaso-motor nerves of the liver have been previously divided. The opinion of Schiff is still further confirmed by the fact that injuries of the vaso-motor conducting paths, in their passage downwards from the medulla oblongata through the cord, are followed by diabetes. Diabetes is also caused by section of the spinal cord anywhere down to the level of the first lumbar vertebra (Schiff, Eckhard), by destruction of the upper and lower cervical, and of the upper thoracic sympathetic ganglia (Eckhard, Cyon), or even after section of the large peripheral nerve trunks, such as the sciatic (Schiff). If the pneumogastric nerve be cut in the neck, stimulation of the upper end is followed by dilatation of the vessels of the liver, and the appearance of sugar in the urine. The most reasonable supposition in explanation of these phenomena is that vaso-motor paralysis of the hepatic artery causes dilatation and engorgement of the hepatic vessels, giving rise

to an increased production of sugar. In 1868, Braun drew attention to the frequent presence of sugar in the urine in cases of sciatica, and this observation has since been confirmed by Rosenstein, Eulenburg, and others.

§ 102. *Neurotic Enlargement of the Spleen and Liver.*—Certain forms of enlargement of the spleen and liver are probably due to paralysis of the vaso-motor nerves of the respective organs. The nerves of the spleen are derived from the semilunar and splenic plexuses, and these are now proved to consist both of afferent and efferent fibres. Section of the efferent fibres causes enlargement of the spleen, along with a dark blue colour of its tissue, while irritation of these fibres causes a reduction of the size of the organ, its tissue becoming of a grey colour from anæmia. The number of the white corpuscles conveyed from the spleen by the veins is diminished in both cases, but the diminution is to a greater degree in vaso-motor paralysis. When, therefore, the spleen has been previously swelled, contraction of its substance leads to a considerable increase of the white blood corpuscles in the veins. The centripetal fibres are, according to Bulgak, found exclusively in the greater splanchnics, while their reflex centre is found in the cord between the first and fourth cervical vertebræ. Strong irritation of the central ends of the vagus and sympathetic in the neck produces reflex contraction of the vessels of the spleen, but appears to be a consequence or phenomenon of commencing asphyxia.

How far enlargement of the spleen in intermittent fever and other malarious diseases and in leucocythæmia is dependent upon vaso-motor action is doubtful. With respect to the liver, it has been found that destruction or extirpation of the cœliac and mesenteric plexuses causes, besides other phenomena, congestion and enlargement of the organ. The liver is also increased in size and congested in cases of diabetes mellitus, caused by paralysis of the vaso-motor nerves of the liver in their cerebro-spinal or peripheral course. It has not yet been decided how far many other kinds of enlargement of the organ are dependent upon vaso-motor action, but it is at least very probable, that the congestion which takes place in frequently repeated attacks of migraine is of this nature.



## CHAPTER VI.

## III.—TROPHONEUROSES.

THE nutritive affections which are caused by disease of the nervous system may, in addition to the anatomical subdivision already described, be subdivided into diseases of peripheral, spinal, or cerebral origin, according as the peripheral nerves, the spinal cord, or the brain is the seat of the lesion.

## (I.)—TROPIC AFFECTIONS OF THE NERVOUS SYSTEM.

*Peripheral Nerves.*—In paralyses of cerebral origin, with the exception of those resulting from disease of the pons, in various spinal paralyses, in all hysterical and many forms of peripheral paralyses, the affected nerves do not undergo any histological changes for a long time. When the paralysis has existed for many years the nerve undergoes slight atrophy, the result of inactivity, or the neurilemma may undergo a moderate degree of hypertrophy, especially when there is coincident muscular contracture.

In some forms of spinal paralyses, atrophy and attenuation of the nerve fibres may be observed, the interstitial tissue being somewhat hypertrophied.

In a third series of cases, of which infantile and traumatic paralysis may be taken as the type, remarkable changes occur in the nerves—changes which have been carefully studied by means of experiments on animals. Nasse was the first who studied those which occur in the peripheral segment of a divided nerve; but A. Waller, as will be hereafter seen, made the most important discovery with respect to the influences which determine nerve degeneration.

§ 103. *Wallerian Degeneration*.—The changes undergone by nerves after section have been studied by Waller, Schiff, Philippeaux and Vulpian, Neumann, Erb, and many others, but the most elaborate account of these changes is given by Ranvier, in his recent work on the histology of the nervous system, and the following summary is mainly derived from that work.

The earlier changes which occur after section of a nerve are limited to the peripheral and central ends of the divided fibre, and do not usually extend, in either direction, beyond the first node of Ranvier, although slight changes may occasionally be observed as far as the second node from the point of injury. If the ends of the divided nerve be examined a few hours after section, the medullary sheath is seen to be swollen and opaque; while the interannular nuclei may already begin to manifest traces of constriction prior to division. Between the divided ends of the nerve there is an accumulation of red and white blood corpuscles. The white corpuscles in the course of a few days form knob-like projections at each end of the nerve, which soon become fused where the loss of substance in the nerve has been small. The white corpuscles after a time increase in size, become spindle-shaped, and ultimately transformed into connective tissue fibres. The divided nerve becomes thus soldered together by means of connective tissue, but this tissue does not take an active part in the regeneration of the nerve fibres themselves.

If, however, the peripheral end of the nerve undergoes paralytic degeneration, a fresh series of changes occur, these taking place almost simultaneously in the entire length of the peripheral portion of the fibre. When the peripheral portion of the divided nerve is examined two days after section, the medulla of the divided fibres is found coagulated, opaque, granular, and broken up into cylindrical masses. The condition of the peripheral fibres of the sciatic nerve of a hare fifty hours after section is represented (*Fig. 14, 1, 2*). The nucleus (*n*) of the interannular segment has become more voluminous and contains a large and well marked nucleolus. The protoplasm which surrounds the nucleus becomes so abundant and well developed at the level of the nucleus, that it fills the calibre of the nerve tube and completely interrupts the medullary sheath. Accumulations of protoplasm also take place at other points of the interannular segment, and these may constrict, more or less deeply, the medullary sheath, or may even completely interrupt it. The protoplasm becomes filled with the fine fat granules into which the myeline has been converted, and a similar granular *debris* may be observed outside the sheath of Schwann, and in the substance of the cells of the endoneurium. During the next two or three days the segmentation of the medullary sheath proceeds, and the cylindrical masses become broken up into globular masses (*Fig. 14, 5 and 6*), which at the end of the first



week after section are converted into drops of variable size, amongst which a progressively increasing number of fine fat granules may be observed. During this period the altered medulla occupies a larger space than in health, so that the fibres appear broader than usual, although their outline is somewhat irregular and wavy. But as the change advances the medulla becomes gradually converted into fat granules, which are absorbed.

It has been asserted by Erb and other authorities that the axis-cylinder persists without apparent injury long after the medullary sheath has disappeared. Ranvier, however, affirms that the protoplasm collects at the level of the interannular nucleus to such an extent that after having pressed upon and absorbed the medullary sheath it attacks and intersects the axis-cylinder (*Fig. 14, 3, p, cy*). The axis-cylinder may also be subsequently cut across by the accumulation of protoplasm at other levels than that of the interannular nucleus.

Subsequently to the fourth day after section a new phenomenon may be noticed. The nucleus may be observed situated near the middle of an interannular segment, and containing a very large and distinct nucleolus (*Fig. 14, 5, n*). The nucleolus may first be seen to undergo hour-glass contraction, and after successive changes it ends by dividing into two. After a time the nucleus exhibits similar transformations, and ends by becoming completely subdivided into two nuclei, each of which may subsequently undergo subdivision. In *Fig. 14 (6, n'' n'' n'' n'')*, four nuclei are observed to correspond to one interannular segment. At a later period of the degenerative process the greater portion of the medulla is absorbed, although some globular masses may accumulate at certain points in the length of the fibre (*Fig. 14, 6, m*); the process of multiplication of nuclei ceases; and even the axis-cylinder disappears from considerable portions of the length of the fibre. The result of this process is that the sheath of Schwann is completely empty of contents at certain points, and collapses so that the degenerated fibre appears exceedingly slender (*Fig. 14, 9, a a*). The calibre of the tube is distended at intervals by elongated nuclei arranged in a series (*Fig. 14, 9, n n*), by fragments of the axis-cylinder, or by globular masses of altered myeline (*Fig. 14, 9, m*); so that the degenerated nerve tube appears as a delicate pale band with irregularly undulating contour.

With the disappearance of the medullary sheath the degenerated nerve loses its white colour, and assumes a grey appearance, the fibres shrink, and the nerve looks small and wasted. It is probable that this process is accompanied by proliferation of the cells of the endoneurium and even of the perineurium; a process which is followed, in long-standing cases, by cicatricial shrinking or cirrhosis, rendering denser the texture of the degenerated nerve, but adding still further to its atrophied appearance.

#### § 104. *Regeneration of Nerves.*

The process of regeneration differs greatly according as the nerve has been simply divided by a sharp knife or a portion has been excised. In





both cases the portion above the point of division remains normal, with the exception of the changes which occur in the medullary sheath as far as the first node of Ranvier. If the ends of the divided nerve are maintained in apposition during the reparation process, it is probable that the axis-cylinders of the central may become united with those of the peripheral end before any serious degenerative changes have occurred in the latter, and that in this manner "union by first intention" is obtained. The case is, however, different when the ends of the nerve are not maintained in apposition, and when a portion of the nerve has been excised. Waller, who was the first to draw attention to the importance of this subject, thought that the nerve tubes of the peripheral segment degenerated in their entire extent, and that the regenerative process was due to an active growth of the nerve tubes of the central segment. Remak subsequently described the formation of new nerve fibres in the interior of the degenerated nerve tubes of the peripheral segment. The delicate investigations of Ranvier have, however, shown that the axis-cylinder is completely destroyed in the peripheric segment, and that the active growth of new fibres proceeds from the nerve tubes of the central segment. Ranvier describes several ways in which the central ends give rise to new nerve fibres; but it must suffice to mention here one or two of the more common of them. The central tube

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granules and drops of myeline (*g* and *m y*) may be observed. The medullary sheath is completely interrupted at the level of the nucleus, while at *a* it has undergone strangulation.

3. Appearance presented by the peripheral fibres four days after section of the sciatic nerve of a hare, originally hardened in a solution of bichromate of ammonia and stained by picocarmine; (*c y*) fragments of the axis-cylinder retracted, somewhat tortuous and embedded in a mass of myeline (*m y*); (*p*) protoplasm swollen and granular.

4. Fibre same as 3, but originally coloured by picocarmine after maceration in perosmic acid; (*n*) nucleus compressing and partially interrupting the medullary sheath and the axis-cylinder; *p*, protoplasm.

5 and 6.—Fibres from the peripheric portion of the sciatic nerve of a pigeon three days after section (same method of preparation as 4). 5. Median portion of an interannular segment presenting a single swollen nucleus (*n*) surrounded by a mass of protoplasm (*p*). 6. Presents four nuclei (*n" n" n" n"*) in a single interannular segment. The protoplasm (*p*) which surrounds them is not segmented, but contains masses of myeline in its interior.

7. Fibres from the central end of the sciatic nerve of a hare ninety days after section (same method of preparation as 4). Dark upper portion represents primitive nerve fibre surrounded by the sheath of Schwann (*s*), and terminating by a knobby enlargement of its medullary sheath (*b*). From the extremity of this termination a second tube (*t'*) issues, which divides and subdivides until it forms a bundle of very fine medullary fibres (*F*), surrounded by a secondary sheath (*s'*) emanating from the sheath of Schwann; *m*, drops of myeline derived from the old nerve fibre.

8. A large nerve fibre of the central extremity of the pneumogastric nerve of a hare seventy-two days after section—maceration in perosmic acid. The medullary sheath (*t*) terminates by a knobby extremity (*b*), and from this extremity secondary medullated nerve tubes (*t' t'*) issue, as well as fibres without myeline; (*s*) the sheath of Schwann of the primary fibre forming secondary nerve sheath (*s'*) for the nerve fibres which issue from it.

9. A nerve tube of the peripheric segment of the pneumogastric of a hare six days after section. The portions *a a*, which are neither occupied by drops of myeline nor by nuclei, are collapsed, and the tube is contracted at this level, *a n*, nuclei of the interannular segment, having undergone proliferation; *m m*, drops of myeline.

terminates by a slight enlargement at one of the nodes (*Fig. 14, 7*), and from this extremity a nerve tube (*t'*) issues, which, although thin, is characterised by a medullary sheath (*s'*) and interannular nucleus. This tube soon subdivides into two others of almost the same size as itself, and each of these in their turn subdivides into two new nerve tubes, so that the old sheath of Schwann becomes distended by a bundle of new fibres (*Fig. 14, 7, F*). Rounded masses of altered myeline (*m*) are often observed at intervals to lie between the old sheath and the young fibres.

At other times several nerve tubes (*Fig. 14, 8, t' t' t'*), some of them possessing distinct medullary sheaths, while others consist of naked axis-cylinders, issue from the extremity of the central fibre, and these also extend towards the periphery. These new fibres on reaching the peripheral segment penetrate for the most part into the interior of the degenerated tubes; but some of them, according to Ranvier, insinuate themselves between the old sheath and the substance of the endoneurium. It would appear, therefore, that Waller's original opinion with respect to the centrifugal development of the new nerve tubes from the tubes which are still connected with the ganglion cells of the anterior horns is confirmed by the elaborate researches of Ranvier, whatever may be the details of the successive steps by which the union is effected. The duration of the process of regeneration varies according as there is simple division of the nerve or a portion is resected, and is also affected by numerous other circumstances. Schiff found complete reunion of divided nerves in young animals in from seven to fourteen days, and Paget found clinically in two cases traces of returning sensibility in fifteen days. After division of the facial nerve paralysis of the facial muscles usually diminishes only after two or three months. Sensory functions are restored considerably sooner than motor functions. If the portion of nerve destroyed or resected exceeds a certain length regeneration will not take place. Regeneration is not likely to take place when the length of lost nerve exceeds two inches, but Weir Mitchell states that in man there are instances of at least three having been restored.

§ 105. *Degeneration of the Conducting Paths of the Spinal Cord and Brain.*—It has been found that, when the conducting paths of the spinal cord and brain are interrupted in any part of their course, the fibres on one side of the lesion undergo degeneration until their termination in grey substance. It is evident, therefore, that their degeneration is only of much importance when the fibres of the conducting path possess considerable length. When the fibres are, after a short course, interrupted by cells, the degeneration which follows injury will be limited to the immediate neighbourhood of the lesion; but when the fibres pursue a long course uninterrupted by



grey matter, the degeneration may extend for a long distance beyond the seat of injury.

It is unnecessary to describe in detail the process of degeneration as it occurs in the fibres of the central conducting paths, inasmuch as it is in all essential particulars similar to that which occurs in the peripheral segments of divided nerves. About three weeks after the injury the fibres of the conducting path become degenerated in their entire length, the conducting path itself assumes a grey or yellowish grey colour, and becomes somewhat denser than the surrounding healthy white substance, hence the process is called *sclerosis*. It has also been observed that the fibres of some of the conducting paths of the encephalo-spinal system undergo degeneration or sclerosis above the seat of the lesion, while others degenerate below the seat of lesion. If the continuity of the spinal cord, for instance, be interrupted, say, in the upper dorsal region, by the pressure of a tumour, or by Potts' disease of the vertebral column, and death supervene a few weeks afterwards, the columns of Goll (*Fig. 3, g*) degenerate above the seat of the lesion as far as the clavate nucleus (*Fig. 3, c n*) and the direct cerebellar fibres (*Fig. 3, d c*), probably until their termination in the grey cortex of the cerebellum, although the degeneration has not been actually traced beyond the upper end of the medulla oblongata. The fibres of the pyramidal tract (*Fig. 4, p, T*, and *Fig. 2, 55 M*), on the other hand, undergo degeneration below the seat of the lesion down to their terminations in the grey anterior cornu. If the fibres of the pyramidal tract be injured either in the spinal cord, medulla oblongata, pons, middle third of the crusta (*Fig. 5, p' p*), middle third of the posterior half of the internal capsule, corona radiata, or where they join the cortex of the brain, the fibres below the seat of injury undergo degeneration down to their termination in the grey anterior cornu of the spinal cord. Briefly expressed, it is said that the columns of Goll and the direct cerebellar tract undergo *ascending sclerosis*, while the pyramidal tract undergoes *descending sclerosis*.

§ 106. *Theory of Nerve Degeneration*.—Waller was the first to observe that when a mixed nerve is divided the peripheral portion degenerates throughout its whole course in a few weeks, while the portion attached to the cord does not degenerate. He found, however, that when the posterior or afferent root of the nerve is cut between its ganglion and the cord, the peripheral portions attached to the ganglion do not degenerate, but the small portion attached to the cord soon wastes. He therefore concluded that the efferent fibres receive their nutritive influence from the caudate cells of the anterior horns, and the afferent fibres from the ganglion of the posterior roots, and

formulated the general law that nerve fibres degenerate when they are separated from their trophic centres. With respect to the central conducting paths it appears certain that the degeneration occurs along the line of the conduction of the fibres. The columns of Goll and the direct cerebellar tract, which undergo ascending sclerosis, are centripetal, and the pyramidal tracts, which undergo descending sclerosis, are centrifugal conducting paths. It would appear that the large caudate cells found in the fourth layer of the cortex of the brain in the psycho-motor centres, and which are almost in every respect similar to the caudate ganglion-cells of the anterior cornua of the cord, form the trophic centres for the fibres of the pyramidal tract; but whether the cells of the ganglia of the posterior roots, those of the posterior grey horns, or of the vesicular column of Clarke form the trophic centres of the fibres of the columns of Goll, and of the cerebellar tracts, is unknown.

It becomes interesting to know why simple separation of a nerve fibre from its trophic centre should produce the changes which have just been described. These changes, broadly expressed, consist in the progressive destruction of the special elements of the nerve fibre—the medullary sheath and the axis-cylinder—along with increased nutritive activity of the general structures—the nucleus and protoplasm. The process of development, both with respect to structure and function, is always characterised by the subordination of the general to the special, while the process of degeneration manifests a reverse tendency of subordinating the special to the general. The special structures of the nerve fibre, the medullary sheath, and axis-cylinder could never have been developed unless the general functions of the nucleus and protoplasm had been kept to some extent in check by some force, and this check may be supposed to be exercised by a trophic centre. When once the moderating or inhibitory influence of the trophic centre is removed, the special structures lose in the struggle for existence, while the general functions of the nucleus and protoplasm become more active and prominent. It is not necessary, therefore, to suppose that the multiplication of the nuclei and the great increase in the amount of protoplasm present during the degenerative process is the result of irritation; and if multipli-



cation of nuclei in this instance be not a test of previous irritation, it is not a trustworthy test of irritation at other times. The multiplication of the nuclei of muscular fibres, for instance, which takes place during degenerative processes is no more a sign of previous irritation than a similar multiplication in the case of degeneration of nerve fibres.

## (II.)—MUSCULAR TROPHONEUROSES.

In order to gain a better insight into the nutritive changes which muscles suffer in connection with certain diseases of the nervous system, it is necessary to mention briefly the leading characteristics of their healthy structure.

### § 107. *Structure of Healthy Muscle.*

1. *Unstriated Muscles.*—The elements of unstriated muscle are elongated, spindle-shaped cells of variable length, each containing an oblong nucleus (*Fig. 15, 10*). Each muscle cell consists of the following parts (Klein): (*a*) a fine sheath possessing transverse linear thickenings; (*b*) a central bundle of fibrils, representing the contractile substance or core; (*c*) an oblong nucleus, including a fine network which anastomoses at the poles of the nucleus with the bundle of fibrils of the core. The muscle cells are aggregated to form a bundle, the cells being held together by a cement substance in which flattened connective tissue cells, and occasionally a few connective tissue fibres, may be observed, which represent the *endomysium*. The individual bundles are in their turn aggregated to form larger bundles, by means of fibrous connective tissue of the ordinary description, constituting the *perimysium*. The *endomysium* and *perimysium* correspond respectively with the *endoneurium* and *perineurium* of nerves.

2. *Striped Muscles* consist of long cylindrical fibres aggregated so as to form bundles, these again being grouped into larger fasciculi. The bundles are surrounded and separated by ordinary fibrous connective tissue, constituting the *perimysium*. From the latter minute bundles of connective tissue, with connective tissue cell plates, pass between the individual muscle fibres to form the *endomysium*.

When a muscular fibre is examined along its longitudinal axis it shows (*a*) transverse broad thin bands of a highly refractive substance (*Fig. 15, 4, b*), and (*b*) narrow bright bands of a less refractive substance (*Fig. 15, 4, d*). The dim band alone constitutes the contractile portion of the fibre, and it may therefore be called the *contractile disc*; while the bright band represents interstitial substance, and may be called the *interstitial disc*. The *contractile disc* is, however, not a simple, but a very compound body. The disc is composed of *sarcous elements*, each of which consists of a prismatic corpuscle. These prismatic corpuscles

FIG. 15.

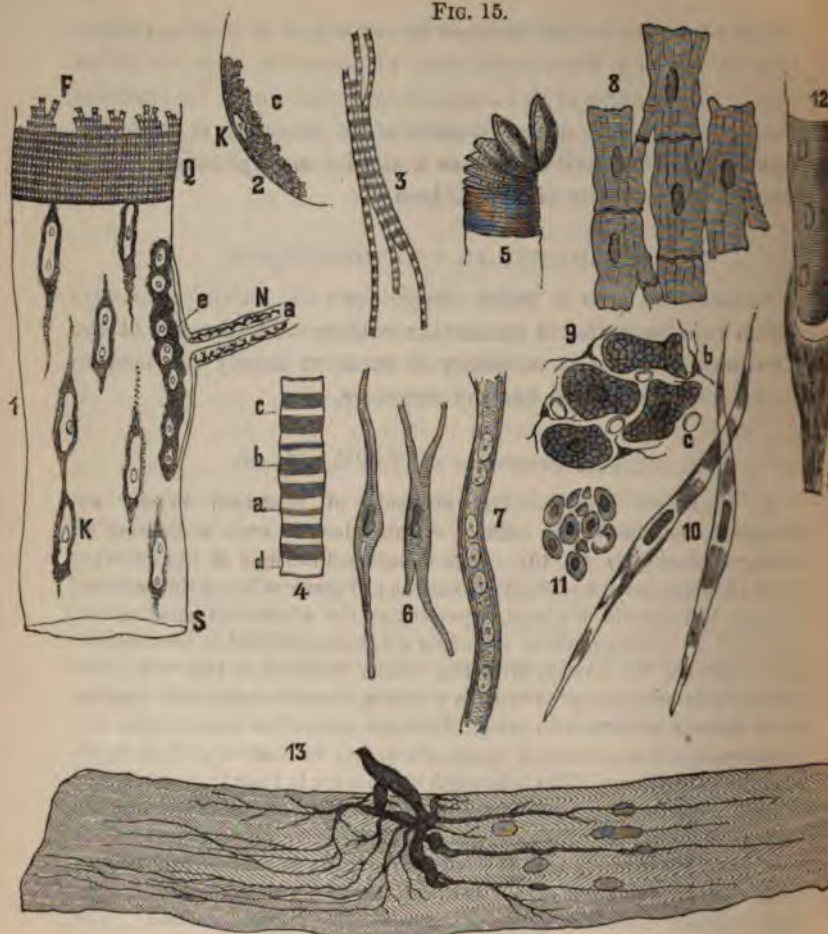


FIG. 15. *Structure of Muscle* (from Landois' "Physiologie").—1, Schema of the different parts of a Striated Muscular Fibre; S, Sarcolemma; Q, Transverse Striation; F, Fibrillæ, the presence of which give rise to the Longitudinal Striation; K, Nuclei of Muscular Fibre; N, Termination of the Motor Nerve, showing (a) the Axis Cylinder, which terminates in Kuhne's motor end-plate. The latter is viewed in profile, and rests on a layer of nucleated protoplasm. 2, Schema of a portion of a Transverse Section of a Nerve Fibre, showing c, Cohnheim's fields, and K, Nucleus lying under the Sarcolemma. 3, Isolated Fibrillæ from a Striated Muscular Fibre. 4, Portion of a Fibrilla of the Muscle of an Insect (greatly magnified); a, Krause's Lines or Membranes which limit the muscular compartments; b, the dark Doubly Refractive Substance; c, Hensen's Lines; d, the Singly Refractive Substance. 5, Striated Muscular Fibre breaking up into Transverse Discs. 6, Striated Muscular Fibre-cells from the Heart of the Frog. 7, Structure of a Muscular Fibre from a Human Embryo of three months. 8, Longitudinal Section of the Muscle of the Heart. 9, Transverse Section of the Muscle of the Heart; c, Capillaries; b, Connective Tissue Corpuscles. 10, Unstriated Muscular Fibres. 11, Transverse Section of Unstriated Muscular Fibres. 12, Striated Muscular Fibre uniting with its Tendon, S. 13, Interfibrillary Muscular Nerve Fibres in Striated Muscle (after Gerlach).



are arranged side by side, so that when the contractile disc is examined in transverse section they are viewed end wise and present polygonal surfaces (Cohnheim's fields, *Fig. 15, 2, c*). The sarcous elements shrink after death and under the action of various reagents, and they then appear to be embedded in or surrounded by an interstitial substance, having the same chemical and physical characteristics as the interstitial discs. But the muscular fibre can be analysed longitudinally as well as transversely. The fibre can be separated into fibrillæ (*Fig. 15, 3 and 1 F*), which consist of several sarcous element prisms, placed end on end, and held together by a prism of the bright substance of the interstitial disc. In addition to the fibrillæ, nuclei (*Fig. 15, 1 K, 2 K*), or muscle corpuscles, may be observed scattered at intervals in the length of the fibre; these representing the cells from which the fibre was originally developed.

These different elements—contractile and interstitial discs, primitive fibrillæ, and nuclei—are held together so as to form a fibre by some important structures.

The first of these consist of a transparent, structureless, elastic sheath, which surrounds the contents of the muscle fibre like a cuticle, and is called the *Sarcolemma* (*Fig. 15, 1 s*). This sheath corresponds to the *neurilemma* of a nerve fibre. Thin elastic membranous septa pass transversely through the muscular fibre at regular intervals, dividing it into cylindrical compartments—the *muscle-compartments* of Krause. Each of these septa passes through the centre of an interstitial disc (*Fig. 15, 4 a*), so that each compartment holds the contractile disc in its centre, and a thin interstitial disc at each end. According to Hensen a thin transparent "median disc" (*Fig. 15, 4 c*) divides the contractile disc into two, and this occupies the centre of the cylindrical compartment; but this appearance is found only under exceptional conditions (Klein).

*Development of Muscular Fibre.*—Elongated spindle-shaped cells are transformed into striped muscle fibres at an early period of embryonic life. The spindle-shaped cells increase in size, their nuclei undergo repeated division, and the cell substance, beginning from the periphery, becomes differentiated into sarcous elements and interstitial substance (*Fig. 15, 7*). What remains of the original protoplasm around the nuclei represents a muscle corpuscle.

*Termination of Nerves in Striped Muscle Fibres.*—Small nerve bundles situated in the connective tissue of the perimysium form a plexus around the muscular bundles, called the *ground plexus* (Klein). Small groups of nerve fibres come off from this plexus, and pass into the substance of the muscular bundles. They also form a plexus in the substance of the endomysium, called the *intermediary plexus*. From the intermediate plexus isolated medullated nerve fibres (*Fig. 15, 1 n*) enter the individual muscle fibres in an oblique or vertical manner, the sheath of Schwann of the nerve fibre becoming fused with the sarcolemma, while the axis-cylinder (*Fig. 13, 1 a*), having lost its medullary sheath, passes within the sarcolemma. The axis-cylinder branches into several thin fibres,

which form a network with one another (*Fig. 15, 13*) between the fibrillæ but near the surface of the fibre. These fibres lie embedded in a granular, plate-like mass, containing numerous oval nuclei, and termed the nerve end-plate of Kühne.

§ 108. *Histological Changes in Paralysed Muscles.*—The histological changes which occur in paralysed muscles vary greatly according to the nature of the paralysis. Paralysis resulting from disease of the cortex of the brain, or of the centrifugal conducting paths which connect it with the spinal cord, is not followed by active atrophy of the paralysed muscles, unless, indeed, the motor ganglion-cells of the central grey tube become secondarily implicated in the disease. When simple voluntary paralysis has existed for many years, the fibres of the paralysed muscles atrophy to some extent, owing to their long inactivity. This is, however, very different from the active atrophy which occurs when the motor ganglion-cells of the central grey tube, or the efferent fibres which connect these with the muscles, are injured or diseased. Neurotic muscular atrophy may be subdivided for practical purposes into the following stages:—1, Simple atrophy; 2, Atrophy with nuclear proliferation; 3, Cirrhosis of muscle.

1. *Simple Atrophy.*—Less is known with regard to the microscopical appearances of the muscles during the first stage of the disease than in the later stages. In simple atrophy and the early stages of the other forms of atrophy, the muscular fibres undergo a simple diminution in size, without any degenerative changes. A microscopical examination reveals a great number of fibres of small diameter, which preserve their normal striation, and present no trace of fatty degeneration. Both the longitudinal and transverse striation are at times as well preserved as in health (*Fig. 16, a*). It would appear that there is a diminution of the number of fibrillæ of which the fibre consists; while the fibrillæ which remain do not seem to be sensibly diminished in size. At other times the striation becomes more delicate and less marked than in health, probably owing to a diminution in the length of the sarcous elements of the contractile discs (*Fig. 16, b*). The substance of the contractile discs may also present a finely granular aspect, which appears to be the first indication of the more profound chemical change which this substance subsequently undergoes. Even at a very early period of the atrophy indications of proliferation of the muscle corpuscles and of the nuclei of the endomysium may be observed; but these appearances will be best described along with the second stage of the process. If the connection between the muscle



and the ganglion-cells of the anterior grey horns of the spinal cord be now restored, the atrophied muscular fibres gradually resume their normal size and complete restoration takes place.

2. *Atrophy with Nuclear Proliferation*.—When the muscles are examined from three to five weeks after the occurrence of the injury, it is found that, in addition to the changes already described as occurring during the first period, the contents of the muscular fibres have undergone a profound degeneration. The finely granular appearance of the contents of the fibre, which was mentioned as being with difficulty observed during the earliest stage, now becomes a characteristic feature of the process (*Fig. 16, c*). The granules at first probably consist of altered protein, and are soluble in acetic acid and insoluble in ether; but they soon become distinctly fatty, being insoluble in acetic acid and soluble in ether. The primitive fibrils now disappear, and only small fragments of the fibres present here and there either transverse or longitudinal striation. But the most remarkable change which occurs in this stage of muscular atrophy consists of proliferation of the nuclei or of the muscle corpuscles. At first the corpuscles are observed to be more numerous than in healthy fibres, but in the later stages of the process the sarcolemma may become almost filled with masses of nuclei surrounded by granular and fatty detritus, while the contents of the fibre are completely disintegrated (*Fig. 17, b, b*). During the time in which the muscular fibre is becoming thus altered the nuclei of the endomysium also proliferate, so that a much larger number of those may be observed lying between the atrophied muscular fibres than between healthy fibres; and it is to the subsequent changes which these connective tissue corpuscles undergo that the third stage of atrophy is mainly due.

FIG. 16.



FIG. 16 (after Hayem). *Atrophy of Muscular Fibres from a Case of Infantile Paralysis*.—*a*, Fibres of normal size, showing multiplication of nuclei; *b*, simple atrophy, with granular degeneration; *c*, advanced granular degeneration, with atrophy.

3. *Cirrhosis of Muscle*.—The connective tissue corpuscles, now greatly increased in number, elongate into fibres which form narrow bands of fibrous tissue running parallel to the direction of the muscular fibres, and cicatricial contraction of which gives rise to organic shortening of the muscle, or rather of the fibrous tissue, which has now replaced the muscular tissue. When this condition has been reached the muscular fibres become destroyed, and restitution of the muscle is impossible. The bands of fibrous tissue which result from the development into fibres of the cells of the endomysium, and, indeed, it might be added of the cells of the perimysium, for the process extends to them, contain many oat-shaped nuclei and connective tissue cells (*Fig. 17, a, a*). The cells not unfrequently become distended with fat, and the deposit of fat in the interstitial tissue may be so abundant that the original volume of the muscle may be maintained or even exceeded.

These, then, are the outlines of the leading features presented by the different degrees of neurotic muscular atrophy, although the process varies considerably under different circumstances. Some of these varia-

FIG. 17.

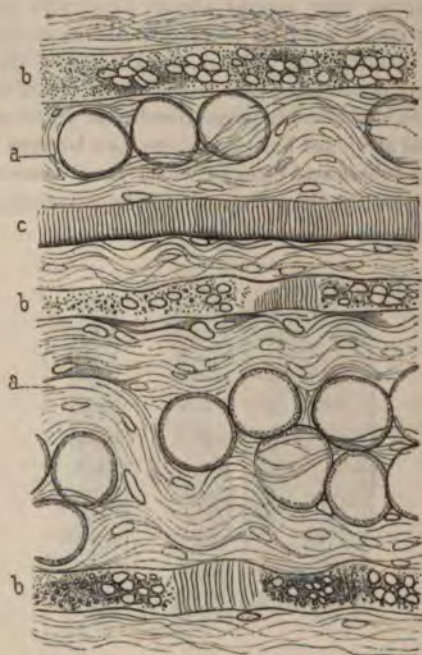


FIG. 17 (after Hayem). *Infantile Paralysis*.—*a a*, excess of connective tissue, containing a large number of connective tissue and fat cells; *b b*, atrophied muscular fibres, containing a large number of nuclei; *c*, simple atrophy of muscular fibre.



tions will be described in the special part of this work. It must suffice at present to point out, that the first stage, or simple atrophy, is met with in those cases where the paralysed muscles exhibit the first degree of the reaction of degeneration; the second stage, or atrophy with muscular proliferation, where the muscles exhibit the second degree of this reaction; and the third stage, or cirrhosis of the muscle, where the affected muscles exhibit the third degree of the reaction of degeneration. Electrical examination will therefore enable us to determine the degree of muscular degeneration which has been reached, and consequently to foretell whether the patient will make a speedy recovery, or a slow, tedious, and imperfect one, or whether the paralysis is completely incurable.

§ 109. *Theory of Muscular Atrophy.*—It is now fully ascertained that active neurotic atrophy of muscular fibres, with rapid loss of faradic contractility of the affected muscles, never occurs except when the spinal motor mechanism is injured, either by disease of the ganglion cells of the anterior horns of the spinal cord or of the corresponding cells in the medulla oblongata, or of the efferent fibres which connect these with the muscles. Active neurotic atrophy is found associated with acute diseases of the grey substance of the cord, as acute central myelitis, spinal apoplexy, fractures and luxations of the vertebral column, infantile spinal paralysis and related diseases. It occurs also in connection with diseases of the efferent fibres, either in their course through the anterior columns or at their origin in the anterior roots (*Fig 11, a a*), or in their course to the muscles through the peripheral nerves.

The question now arises as to the nature of the injury from which active atrophy results. It was first stated by Brown-Séquard that nerve-irritation alone is capable of determining that rapid and early atrophy of the muscles, which is accompanied by decrease or disappearance of faradic contractility; and that simple section of a nerve does not induce atrophy and loss of electrical reaction until a considerable time, often many years, has elapsed. Under the latter circumstances the atrophy results from prolonged inactivity. The observations of Erb and Ziemssen appeared to contradict this theory, but the acute criticism to which Charcot has subjected these experiments seems to show that the opinion of Brown-Séquard must still be maintained. The affections of the spinal cord which are associated with active muscular atrophy are, according to

Charcot, all acute diseases, and such as are likely to give rise to considerable irritation; while the diseases of the peripheral nerves, in which muscular atrophy occurs, are almost always of traumatic origin, such as concussion, contusion, compression, imperfect division—all of them calculated to provoke irritative action. When a lesion of a mixed nerve gives rise to active muscular atrophy, the affection is accompanied not by anæsthesia of the territory of distribution of the sensory fibres, but by more or less acute pains, or by various abnormal sensations, with which glossy skin, herpes, and other trophic cutaneous affections are apt to be associated. The presence of these sensory disturbances shows that the afferent fibres of the nerve are in a state of irritation, and this affords grounds for inferring that the efferent fibres are in a similar condition, and not simply divided. This question must, however, be regarded as still an open one, to be decided by future observations and researches.

### (III)—CUTANEOUS TROPHIC AFFECTIONS.

§ 110. *Erythema and Related Conditions*.—Erythema is probably not unfrequently caused by a functional disturbance of the vaso-motor or trophic cutaneous nerves. After traumatic lesions of the peripheral nerves circumscribed cutaneous red patches are often observed on the extremities, resembling chilblains, sometimes associated with a pseudo-phlegmonous swelling of the subcutaneous cellular tissue (Hamilton). Transitory and recurring patches of erythema, which are no doubt of nervous origin, have been observed on the forehead and root of the nose in connection with trigeminal neuralgia, and occasionally on the hand in cases of brachial neuralgia. Erythema is no doubt frequently of reflex origin, as in those cases which arise in the course of digestive disorders.

*Urticaria*.—It is very probable that the urticaria caused by the stings of insects and nettles, that which arises in the neighbourhood of the puncture in subcutaneous injections, and from various other local irritants, is due to nervous irritation. The suddenness with which urticaria occurs in some persons, after eating shell-fish, oatmeal, and other articles of diet, would appear to indicate that it is due in these cases to reflex nervous irritation.



The nervous origin of urticaria is, however, at times very striking. Charcot mentions the case of a woman suffering from locomotor ataxy, who at every paroxysm of lancinating pains developed enormous patches of urticaria over the parts where the severest pains were felt.

§ 111. *Eczema, Herpes, and other Eruptions.*—Vesicular eruptions are often observed after injury to the nerves. Drs. Weir Mitchell, Morehouse, and Keen have described, under the name of eczematous eruptions, an affection of the skin, consisting of minute vesicles thickly scattered over the tender cutis, or appearing in successive crops of larger vesicles, and occurring after traumatic nerve lesions. These eruptions are limited to the area of distribution of the injured nerve, and are usually associated with severe neuralgic pains; and it is not a little remarkable that these pains often decline on the appearance of the eruption.

Romberg was the first to draw attention to the very frequent association of herpes zoster with neuralgia, and to lay stress upon the analogy of this eruption with the redness and vesicular eruptions which occur in neuralgia, and after injuries of nerves. Herpes zoster is the most familiar inflammatory complication of neuralgia, and its favourite seat is the skin covering one or more of the intercostal spaces. The neuralgia which accompanies herpes zoster generally appears with the eruption; and in aged people it is often exceedingly severe and intractable, and generally continues long after the eruption has disappeared. Mr. Jonathan Hutchinson records several cases of neuralgic herpes zoster of the face, which were attended with iritis, causing serious injury to the affected eye. That herpes zoster is caused by lesions of the spinal cord is shown by its frequent occurrence after traumatic injuries of the vertebral column. H. Schmidt mentions the case of a man who had an attack of herpes zoster in the area of distribution of the first and second lumbar nerves along with neuralgia and anæsthesia three weeks after a severe fall from a ladder. It is, however, possible that the herpes in this case might have been due either to direct injury, or to a descending neuritis of the nerve trunks.

*Spinal, and Cerebral Eruptions.*—Eruptions of zoster frequently occur in chronic myelitis, and especially in tabes dorsalis, and the fact that they are always limited to nerve territories affected with neuralgic and lightning pains, shows that they are undoubtedly of spinal origin. Cases of this kind have been recorded by Charcot, Buzzard, and many other authors. Herpes must probably be regarded as of cerebral origin when the eruption is distributed over almost the whole of one half of the body. An interesting case of this kind was communicated by Oppolzer, in which, after rigors, the left half of the face, and the extremities on the same side, became covered with an eruption of herpes; and although no vesicles formed on the trunk, yet the left half exhibited at the beginning a diffused erythematous blush. Dr. Duncan mentions the case of an aged woman attacked with hemiplegia, in whom an eruption of herpes appeared on the thigh of the affected side almost simultaneously with the motor paralysis. In the case of a child, recorded by Dr. Payne, the eruption of herpes appeared in the territory of the superior branches of the anterior crural nerve three days after an attack of hemiplegia affecting the same side. Charcot relates the case of a young soldier, twenty-two years of age, who was simultaneously attacked with hemiplegia and a vesicular eruption in the inferior extremity of the paralysed side, where it followed the distribution of the superficial twigs of the cutaneous peroneal branch of the musculo-cutaneous nerve. At the post-mortem examination it was proved that the hemiplegia was caused by a focus of softening determined by embolus of a posterior cerebral artery; but the herpes was produced by a totally different mechanism. A spinal arterial branch, probably arising from one of the lateral sacral arteries, was also found obliterated by a blood-clot, forming a comparatively voluminous cord, and adhering to one of the posterior spinal roots of the cauda equina. Charcot thinks that this artery, distended by the embolus, had compressed one of the spinal ganglia, or an initial branch of the sciatic nerve; so that the herpetic eruption in this case would really be of peripheral and not of cerebral origin. This case throws considerable doubt on the cerebral origin of the other cases described, especially as the phenomena observed were not checked by careful post-mortem examinations.



*Pemphigus bullæ* may also develop with great rapidity over various parts of the surface supplied by the cutaneous branches of an injured nerve; and these, according to Charcot, almost always leave after them indelible scars.

Bullous eruptions are also of spinal origin, as in a case observed by Balmer, where an attack of pemphigus occurred in the course of progressive muscular atrophy. In another remarkable case, communicated by Déjerine, the arms and legs became covered by a pemphigus eruption ten or twelve days before death in a case of symmetrical sclerosis of the lateral columns of the cord. Besides the characteristic post-mortem appearances in the cord, the cutaneous nerves underlying the bullæ were found to have undergone degenerative changes. Several cases of pemphigus in connection with spinal disease have been recorded by Chovstek.

That pemphigus is sometimes caused by cerebral lesions is rendered probable by several cases. In one observed by Hesselink, a pemphigus eruption appeared after an apoplectic attack, and disappeared along with the hemiplegia. In another, communicated by Chovstek, bullæ appeared on the outer edge of the paralysed foot on the fourth week after an attack of right-sided hemiplegia, which was preceded by neuralgic pains. The post-mortem examination showed that the hemiplegia was due to a hæmorrhage in the left temporal lobe and lenticular nucleus. The occurrence of a bullous eruption in the early stages of acute bed-sores also shows the occasional dependence of the affection on spinal and cerebral lesions.

Papular and lichenoid eruptions are sometimes caused by lesions of the nervous system. Charcot states that it is not rare to see the skin of the legs and thighs temporarily covered with a papular eruption during paroxysms of the lancinating pains characteristic of locomotor ataxy. Pustular eruptions analagous to ecthyma, and leading to deep ulcerations, may also be developed during the paroxysms of the lightning pains.

§ 112. *Glossy Skin*.—The affection which has been termed "glossy skin" was first described by Paget, who justly regarded the affection as "a sign of peculiarly impaired nutrition and circulation due to injury of nerves." "In well-marked cases," says Paget, "the fingers which are affected (for this appearance

may be confined to one or two of them) are usually, tapering, smooth, hairless, almost void of wrinkles, glossy, pink or ruddy, or blotched as with permanent chilblains. They are commonly also very painful, especially on motion, and pain often extends from them up the arm." Drs. Weir Mitchell, Morehouse, and Keen also drew special attention to this condition, which was frequently observed by them in connection with traumatic lesions of nerve trunks. According to the observations of these authors, when a single nerve, as the ulnar, is injured, this condition of skin is observed in the fingers or toes; but in instances of more extensive nerve injury the palm of the hand often becomes affected, but it is rare for the dorsum to be implicated. In the foot, on the other hand, the reverse condition obtains, the dorsum being the part most liable to suffer. "Glossy skin" appears to be, as Charcot remarks, due to a peculiar inflammation of skin analogous to the condition known as scleroderma. Severe pain, sometimes of a peculiar burning character, is found associated with this morbid condition of the skin.

§ 113. *Alteration in the Pigmentation of the Skin—Partial Leucoderma—Bronzed Skin, Nævi Pigmentosi.*—In partial leucoderma circular patches of the skin are deficient in pigment, giving a white appearance to the affected part, which is more striking by contrast in the coloured races. In these patches the hairs are often white, and cutaneous sensibility is blunted. Patches of leucoderma are sometimes observed on the face in trigeminal neuralgia, and in unilateral facial atrophy. Similar patches have been observed by Eulenburg after injuries to the larger nerve trunks. The affection has also been observed to arise after severe emotional disturbance (Beigel), or in connection with central affections of the nervous system, as locomotor ataxy (Bulkley), and exophthalmic goitre (Raynaud), which prove the frequent dependence of the affection on disease of some part of the nervous system. Increase of pigment of the skin, either partial or diffused, also appears often to depend on nervous disease. The bronzing of the skin in Addison's disease is probably due primarily to an affection of the nervous apparatus. The skin has been observed to become rapidly discoloured after severe emotional distur-



bances; and a case is related by Rostan of a woman under sentence of death, whose skin became in a few days of a dark colour, which gradually disappeared on the sentence being commuted. It is also probable that some of the collections of pigment in the rete called *pigmentary nævi* have a nervous origin.

*Lepra Anæsthetica—Elephantiasis Græcorum.*—The early occurrence and severe degree of anæsthesia in the tubercular variety of leprosy, as well as the manner in which it progresses from the periphery towards the central parts of the affected limbs, would alone indicate that disease of some part of the nervous system is a prominent part of the affection. Characteristic anatomical changes have also been found both in the nerve trunks and in the spinal cord and brain, although it is probable that the alterations in the nerve centres are of a secondary nature.

According to the observations of Virchow the morbid process begins in this form of leprosy, with a perineuritis, causing interstitial cell proliferation to such a degree that the nerve tubes become atrophic through compression. Dr. Vandyke Carter found the brain, spinal cord, and roots of the nerves healthy in ten cases of leprosy; while the nerve trunks, on the other hand, were swollen, dull red, or grey, or semi-translucent, rounded and firm. The morbid changes were found chiefly in the compound trunks, which are situated most superficially, and in the cutaneous nerves just after perforating the deep fascia; the nerves most frequently affected being the supraorbital, great auricular, ulnar, median, and radial. These observations have been confirmed by various other authors. Drs. Danielssen and Böck state that the spinal cord and its membranes are altered. The cord was found indurated, and its grey matter discoloured, yellowish, and devoid of vessels. The membranes were infiltrated with an albuminous deposit, a layer being found between the arachnoid and pia mater. The sheaths of the nerves and the various ganglia were similarly affected. In a case published by Steudener the posterior grey horns of the cord were found softened, and Neumann found in the related disease of leprosy pemphigus thickening of the adventitiæ of the spinal vessels, and the grey substance transformed into a colloid mass. In a case of *lepra mutilans* published by Langhaus, the posterior grey horns of the cord, the columns of Clarke, and the grey commissure were found softened, the morbid changes being especially marked in the upper part of the dorsal region and in the cervical enlargement.

#### § 114. *Bed-sores and Related Conditions.*

*Decubitus Acutus.*—The rapid development of bed-sores in

connection with severe spinal and cerebral lesions has been specially studied by Samuel, and more recently by Charcot; although, indeed, Bright directed attention to this affection many years ago. The bed-sore usually occupies the sacro-gluteal regions, but it may appear on all parts of the trunk and limbs subjected to a somewhat continuous pressure. In some exceptional cases the affection is produced in the entire absence of pressure, or any other recognisable exciting cause.

Some days or even hours after the occurrence of a severe spinal or cerebral lesion, or after a sudden exacerbation of these affections, one or several erythematous patches, of variable extent and irregular form, appear on certain points of the skin. The skin has a rosy hue; sometimes it is dark red or violet, but the colour momentarily disappears on pressure with the finger. Within twenty-four or forty-eight hours the central part of the erythematous patch is covered with vesicles or bullæ, the contents of which, at first colourless and transparent, become more or less opaque, reddish, or brown-coloured. Under favourable circumstances the vesicles may wither, dry up and disappear, and the part recover without further change (Charcot). As a rule, however, the vesicles burst, and leave ill-looking ulcerations, the bases of which are composed of the true skin infiltrated with blood, and usually in a state of phlegmonous inflammation. In such cases the subcutaneous connective tissue, and even the subjacent muscles, are infiltrated with blood. The base of the ulcer soon perishes by gangrene; the neighbouring skin becomes inflamed to a greater and greater extent; and the gangrenous destruction extends deeper and deeper, laying bare and including in its destructive operation, muscles, tendons, fasciæ, ligaments, and even the subjacent bones.

One of the most remarkable characteristics of this affection is the rapidity of its development, the entire cycle of changes being completed in a few days. Cystitis and hæmaturia are not unfrequent complications of this condition, and the muscles often become the subjects of rapid atrophy.

*Sequelæ.*—Such extensive gangrene soon gives rise to a more or less remittent fever, with severe chills, and great variations of temperature. It may also cause *purulent* infection, with



production of metastatic abscesses in the viscera, which are principally observed in the lungs (Charcot and Ball\*). The fatal result in these cases is preceded by general marasmus.

One other complication is worthy of notice. The gangrenous process extends to the sacral bones; and with the destruction of the sacro-coccygeal ligament the vertebral canal is opened, so that the pus and the gangrenous ichor have now ready access to the fatty cellular tissue which surrounds the dura mater; or they may penetrate this membrane, and thus make their way to the cavity of the arachnoid. This grave accident gives rise either to a *simple purulent* or to an *ichorous* ascending meningitis, which rapidly reaches the base of the brain, and leads to a fatal result.

*Simple Chronic Decubitus.*—Chronic bed-sore arises usually in a somewhat different way. In chronic diseases of the spinal cord the portions of skin subjected to pressure in sitting and lying assume a dark red colour, and at times become covered with superficial ulcerations. After a time a black spot appears on the reddened portion of skin, and if the pressure is continued, it enlarges rapidly, and the affected skin dries up into a hard leathery mass. In a short time a boundary line of inflammation forms around the gangrenous portion of skin, and the latter may, under proper treatment, be thrown off, leaving a more or less healthy granulating surface, which, under favourable circumstances, may cicatrise.

But if the pressure be continued, or if the primary disease of the nervous system undergo a fresh exacerbation, the ulcerated surface assumes a dark violet colour, the gangrene spreads rapidly, and all the destructive changes characteristic of acute bed-sore make their appearance, and soon lead to a fatal result (Erb).

This form of gangrene may occur at any place exposed to continued pressure; but its favourite sites are the coccyx and buttocks, the trochanters and ischiatic protuberances, heels, knees, the spinous processes of the vertebræ, the shoulder blades, and elbows.

The exciting causes of bed-sore, besides continuous pressure, are neuro-paralytic hyperæmia, complete immobility of the paralysed parts, absence of sensation, and the irritation caused by discharges from the bladder and

\* Union Médicale, 26 et 28 Janvier, 1860.

rectum. But although these greatly favour the formation of bed-sores, yet they are of themselves, either separately or combined, inadequate to produce the more acute form of the affection; and the latter may form even after every precaution in the way of cleanliness and protection of the parts has been taken to prevent it. It is necessary, therefore, to assume a grave lesion of trophic fibres and centres, as being operative in the formation of acute bed-sore. Acute decubitus is found associated with severe traumatic lesions of the cord, acute myelitis, hæmatomyelia, and it has been observed in unilateral lesions of the cord, not on the paralysed side but on the anæsthetic side. This fact appears to show that the cutaneous trophic fibres decussate in the cord, like the sensory fibres. It is worthy of note that bed-sore does not form in some spinal diseases associated with muscular atrophy, as infantile paralysis. This affection also becomes rapidly developed in some cases of apoplexy from intracranial hæmorrhage or partial softening of the brain; and in such cases the bed-sore does not occupy a median position in the sacral region, as in cases of spinal origin, but forms towards the centre of the gluteal region on the paralysed side. Chronic decubitus is found in chronic transverse myelitis, in the last stages of tabes dorsalis, and also in peripheral paralysis caused by pressure on the cauda equina.

#### § 115. *Trophic Disorders of the Nails and Hair.*

After section of the sciatic nerve in mammalia the limb becomes swollen at its extremity, the toes ulcerate, and the nails fall off. Schröder van der Kolk, who first performed the experiment, attributed the nutritive affection to the loss of the normal nervous action. Brown-Séquard repeated the experiment on guinea-pigs and rabbits, and showed that no notable nutritive changes took place in the paralysed limb, when the extremity was prevented from coming in contact with the hard ground. Traumatic lesions of nerve trunks in man, in which the nerve is not completely divided, are not only often followed by cutaneous nutritive disorders, but also by various deformities of the nails of the fingers or toes, according as the nerves of the upper or lower extremity are affected. In such cases the affected nails become greatly curved—both laterally and longitudinally—furrowed, dry, and cracked at their extremities, and of a yellowish brown colour. These deformities may also occur in connection with neuralgic affections, idiopathic neuritis of sensory nerve trunks. They may also be found in connection with acute and chronic myelitis, or as a result of hemiplegia; but in the latter case it is very probable, as will here-



after be more fully pointed out, that the spinal cord participates in the affection. The hair frequently suffers from nutritive disorders, subsequent to lesions of the nervous system. Such disorders often occur after experimental injuries of nerve trunks in animals. After section of the infraorbital nerve in rabbits the long hair of the beard often falls out, while chemical irritation of the sciatic nerve in the same animal, followed by ascending neuritis and myelitis, is frequently associated with loss of hair on the posterior part of the body (Eulenburg). Local affections of the hair often occur in man after traumatic lesions of nerve trunks, or in connection with idiopathic neuritis and neuralgia. The hairs over the region of distribution of a nerve affected with neuralgia have sometimes been observed to become hypertrophied, and even increased in number; but as a rule the effect of neuralgia upon the hair is to make it brittle, and to cause it to fall out in considerable quantities. Localised greyness of the hair is often associated with ophthalmic neuralgia, and it may also involve that of the eyebrow of the affected side; and, what is a still more remarkable fact, this greyness often assumes an intermittent character, increasing during, and for sometime after, an acute attack of pain, while a partial or total restoration of colour takes place in the interval between the paroxysms (Anstie). Severe emotional disturbance has been known to cause the hair of the head to turn grey suddenly in a single night. After severe injuries of nerve trunks, which give rise to nutritive cutaneous disorders, and especially to "glossy skin," the hair completely disappears from the fingers affected (Weir Mitchell). Occasionally after injuries of nerve trunks, and in chronic myelitis, an increased growth of hair has been observed.

#### § 116. *Cutaneous Secretory Affections.*

Nervous anomalies in the secretion of the skin may occur both in central and peripheral diseases of the nervous system. Various pathological facts appear to prove the existence of cutaneous secretory nerve fibres independently of the vasomotor nerves. Diminution or abolition of the secretion of sweat may at times exist, especially in paralysed extremities, side by

side with the phenomenon of vaso-motor paralysis, such as local increase of temperature and redness; and conversely, increased secretion of sweat often exists on the palms of the hands in connection with vaso-motor contraction and diminution of temperature. In diffused sweats caused by severe emotional disturbances the temperature is diminished, hence these are called "cold sweats." The recent experiments of Luchsinger show that peripheral irritation of the divided sciatic nerve in animals induces an increase of sweat in the paralysed parts.

The cutaneous secretory neuroses consist of excessive sweating or *hyperidrosis*, diminution or absence of the secretion or *anidrosis*; and changes, not in the quantity, but in the quality of the secretion, which may be compendiously grouped under the term *paridrosis*. The profuse sweating of acute disease, as intermittent fever, that which results from the action of various toxic agents, as opium and chloroform, the partial sweats which occur during hysterical and epileptoid attacks, are all undoubtedly of nervous origin. Still more striking examples are to be found in the unilateral perspirations which have been described under the name of *hyperidrosis unilaterialis*. This affection is sometimes limited to one half of the head, and at other times extends to the arm of the same side, or even occupies the entire half of the body, and is usually associated with severe nervous affections, such as hemicrania, Graves's disease, diabetes mellitus, tabes dorsalis, and dementia paralytica. It is probably connected with lesion of the sympathetic or of the cerebro-spinal centres with which the latter is united. Compression of the cervical sympathetic by tumour gives rise to cutaneous redness, contraction of the pupil, and increased perspiration on the same side of the head (Gairdner, Ogle, Verneuil). In a case of unilateral sweating of the head, described by Fränkel, pathological changes consisting of varicose dilatation of the vessels and commencing pigmentary degeneration of the ganglion cells were found by Ebstein in the inferior cervical ganglion of the sympathetic of the same side. Guttmann has published a case in which unilateral sweating of the head was associated not with contraction but dilatation of the pupil of the same side, along with slight exophthalmos. Meyer has shown that by galvanisation of the cervical sympathetic in man an increased secre-



tion of sweat as well as a slight rise of temperature may be induced in the arm of the same side. Nitzelnadel has, on the other hand, found that in a case of hyperidrosis unilaterialis galvanisation of the sympathetic was followed by diminution of the secretion. Przewoski has recently shown that galvanisation of the cervical sympathetic in healthy men influences the circulation both in the half of the head and the arm of the same side, and either increase or diminution of secretion and temperature may occur according to the choice of electrodes and the method of application. Some cases of unilateral hyperidrosis cannot be explained on the supposition of an affection of the cervical sympathetic. In a case described by Pokroffsky\* a profuse perspiration burst out over the right half of the face of the patient always during meals, which appears to have been connected with an old parotitis implicating the right facial nerve, and leading to a slight degree of asymmetry of the face.

*Anidrosis* is a frequent symptom of fevers, diabetes mellitus, chronic Bright's disease, and of certain skin diseases. This condition is also often associated with many profound neuroses, as dementia paralytica. The diminution of perspiration, which is caused by various toxic agents, such as atropine, is evidently due to action on the nervous system. Abnormal dryness of the skin is also observed as a local symptom in association with other nutritive changes. A good example of local dryness of the skin occurs in unilateral atrophy of the face, and a similar local condition may also be found on the extremities in the course of the most different cerebral, spinal, and peripheral chronic nervous affections.

*Paridroses* of the cutaneous secretion due to nervous disease are of various kinds, and no doubt further observations will greatly add to their number. In some nervous affections the secretion manifests a peculiar odour (*Osmidrosis*). The American surgeons observed excessive sweating, with strong odour of vinegar, after severe contusions of peripheral nerves; and in one case the smell resembled that from a bad drain. The secretion at other times becomes changed to a black, blue, red, or green colour (*Chromidrosis*). Coloured perspiration occurs generally in hypochondriacs, in women with uterine disorders

\*Berlin Klin Wochenschrift, 1875, No. 13.

of various kinds; or as the result of grief, fright, and other emotional disturbances. In some few cases extravasation of blood takes place into the sweat glands, giving rise to bloody sweating or *hæmidrosis*. This condition appears occasionally to be vicarious of the menstrual flux (Hebra), but it is usually associated with hysteria and other central nervous affections.

§ 117. *Theory of Cutaneous Trophic Affections.*—Pathological facts appear to warrant the inference that the cutaneous trophic and secretory fibres are distributed to the surface along with the sensory fibres. The central course of these fibres is not easy to determine, but they apparently pass through the intervertebral ganglia and posterior roots to join the posterior horns of the grey matter of the spinal cord. The central and posterior parts of the grey substance of the spinal cord appear to stand in the same relation to the nutrition of the skin that the anterior horns of grey matter do to the nutrition of the muscles and joints. The central course of the cutaneous trophic fibres is unknown, but these fibres cannot be regarded as identical with the cutaneous vaso-motor nerves, since the latter pass out along with the anterior roots to join the sympathetic through the rami-communicantes, and appear to pass through the cord in the antero-lateral columns.

That the cutaneous trophic fibres are associated with the sensory and not with the motor fibres of the peripheral nerves is shown by the facts that these nutritive disturbances are almost always accompanied by severe pains, and other forms of disordered sensation; and that, when the motor and sensory fibres are found in separate nerves, such as the facial and fifth, the nutritive affections are always caused by disease of the sensory nerves. What part, if any, the ganglia of the posterior roots take in the production of peripheral nutritive affections is not known. The cutaneous nutritive affections caused by disease of the spinal cord are also almost always accompanied by severe sensory disturbances. The cutaneous eruptions observed in locomotor ataxy always make their appearance simultaneously with paroxysms of lancinating pains. The anatomical substratum of locomotor ataxy is, as is well known, sclerosis of the posterior root-zones (*Fig. 11, pr*); and Charcot attributes the various sensory disturbances, which accompany the early stages of this disease, to irritation of the *internal radicular fasciculus* (*Fig. 11, pr'*). Charcot thinks that the cutaneous trophic fibres also pass through this bundle, and that these fibres are consequently liable to irritation simultaneously with the other fibres which it contains.



The cutaneous nutritive disorders which accompany acute and chronic myelitis are, when not resulting from implication of the fibres of the internal radicular fasciculus, due to disease of the central grey tube. That these disorders are caused by disease of the posterior or sensory and not of the anterior or motor portion of the grey substance is conclusively shown by the facts that they are entirely absent in infantile paralysis and the other diseases limited to the anterior grey horns; and that not only are they present in diffused diseases of the grey substance, but also that in unilateral affections of the cord the acute bed-sore forms not on the paralysed but on the anæsthetic side. Very little is known in regard to the connection of the spinal cutaneous trophic centres with the brain. It is probable that some of the minor cutaneous eruptions which occur in cerebral disease may be due to vaso-motor irritation, while some of the severer affections are caused by a simultaneous affection of the spinal cord, or of a peripheral nerve. After deducting these, however, a considerable number of cases remain unaccounted for, and it must therefore be supposed that the spinal cutaneous trophic centres are represented by a higher centre in the brain, but whether it is situated in the medulla oblongata, or pons, or in the cortex of the brain is not known.

The next question to be determined is whether the cutaneous nutritive disorders depend upon paralysis or irritation of the trophic fibres and their continuation through the cord and brain. The careful observations of Weir Mitchell, Morehouse, and Keen, of those who suffered from wounds and other injuries of the nerves during the American war, appear to favour the opinion that cutaneous trophic disorders supervene in those cases in which nerves suffer partial injury without being completely severed from their nerve centres. These disturbances arise while the wound is healing; they are often related to an attack of inflammation in or about the wound, and are usually associated with neuralgic and paralgic phenomena, and rarely with complete anæsthesia and paralysis. It must also be remembered that after gun-shot wounds and other severe injuries of nerves the trophic disorders may appear above the level of the wound as well as below it.

The nutritive disorders which accompany disease of the spinal cord also appear to favour the theory of an irritation rather than a paralytic lesion. The cutaneous eruptions which occur in the course of locomotor ataxy are associated with severe paroxysms of lancinating pains; while acute bed-sore is associated with traumatic injuries of the cord, acute central myelitis, and hæmatomyelia; all of them being affections in which the paralytic phenomena are likely to have been preceded by symptoms of irritation. It has also been pointed out by Charcot that many of the symptoms which accompany the formation of acute bed-sore are indeed indicative of irritation, rather than paralysis. The most usual of these symptoms are priapism, clonic convulsions in the paralysed limbs, tonic convulsions coming on in paroxysms, while anæsthesia of the paralysed parts is by no means constant. Post-mortem examination also reveals

in these cases the presence of purulent infiltration and other evidences of an inflammatory process. It is not improbable, however, that chronic decubitus may be caused by paralysis, instead of irritation of trophic centres or fibres, inasmuch as it appears in the terminal periods of chronic spinal affections, and has then the characteristics of a passive rather than an active process. In a case of transverse myelitis from disease of the vertebral column, recently under my care, the formation of a chronic bed-sore was preceded by complete anæsthesia of the inferior extremities, and of the sacro-gluteal region. This, however, only shows that centripetal conduction through the cord was interrupted at the seat of the disease—the middle dorsal region—while it is quite possible that the cutaneous spinal trophic centres of the gluteal region were in a state of irritation. The case, therefore, affords no trustworthy evidence in either direction.

#### (IV.)—NUTRITIVE AFFECTIONS OF THE JOINTS AND BONES.

##### *Articular and Osseous Trophoneuroses.*

Pathological facts appear to show that the trophic fibres of the bones and joints are found in the mixed nerve trunks, and that these issue along with the motor fibres from the anterior cornua, where they are, like the muscular trophic fibres, connected with a group of large caudate cells. It is impossible to say at present in what way these trophic fibres and cells are connected with the brain, but there can be little doubt that such a connection exists.

§ 118. *Affections of Peripheral Origin.*—Affections of the joints are very frequent in connection with traumatic injuries of nerve trunks in which the nerves are not completely divided. It has already been pointed out that it is those partial injuries of the nerves which cause nutritive affections of the skin, hairs, and nails, as well as rapid atrophy of the muscles; and the same is true with respect to the trophic changes of the joints and bones. The nutritive affection of the joints may occur at any time subsequent to the first few days after the injury to the nerve, and consists in a painful swelling, very similar to the condition of the joints in subacute articular rheumatism. This swelling may attack any or all of the articulations of a limb. The affection is distinct from the early swelling due to inflammation about the wound, and it is not a part of the inflammatory œdema, which is a common consequence of wounds. After the acute



stage the tissues about the articulations become hard, and partial ankylosis results, which may ultimately destroy the mobility of the joint (Weir Mitchell). Fischer succeeded in producing experimentally in animals disease of the joints, similar to those observed in man, as the result of disease of the nerves. Swelling and thickening of the bones may occur as the result of injuries to nerves, and this may be followed at a later period, in young people, by decided arrest of the growth of the bone (Eulenburg, Ogle, Blum). These nerve lesions may also be followed by periostitis and subsequent necrosis. In progressive cases of unilateral atrophy of the face, the bones of the face participate to some extent in the wasting.

§ 119. *Osseous Affections of Spinal Origin.*—Attention has recently been drawn by Charcot and his scholars to the great frequency with which nutritive changes occur in joints in central diseases of the nervous system. These affections may be subdivided into two kinds—those which run an acute or sub-acute course, and those which assume a chronic form. The first variety is accompanied by tumefaction, redness, and at times by more or less severe pain. The acute form occurs in connection with Potts' curvature, as a consequence of traumatic lesions (Vigues and Joffroy), and idiopathic myelitis (Gull). The influence of infantile paralysis on the nutrition of the bones of the affected limb is well known, the bones of the diseased extremity being often thinner and shorter than those of the healthy limb. Articular affections have also been observed in progressive muscular atrophy and disseminated sclerosis. The articular affection begins either with or without pain, but the tumefaction and redness may be so marked as to simulate acute rheumatism.

*The Chronic Form* of articular disease due to lesion of the nervous system was first accurately described by Charcot, and it is especially observed in connection with *locomotor ataxia*. The deformities of the joints produced by this affection usually occur in the large joints of the extremities, and more especially in the knee, hip, shoulder, and elbow joints. The affection usually begins about the same time as the symptoms of motor inco-ordination, and its onset is accompanied or preceded by severe paroxysms of lancinating pains.

The symptoms begin suddenly in the absence of any appreciable external cause, generally without any pain or febrile reaction, and the joint may be enormously swelled within twenty-four hours from the commencement of the affection. The general tumefaction disappears after a few days, but a more or less considerable swelling remains, due to the accumulation of serous fluid in the joint, and in the periarticular serous bursæ. One or two weeks after the invasion, the fluid disappears from the joint. In the *benign* form of the affection a complete cure may be obtained, but in the *malignant* form the articular surfaces become greatly altered and roughened, so that cracking sounds are heard on movement. After a time the heads of the bones become atrophied and worn, the ligaments become relaxed, and the surrounding muscles atrophied and enfeebled, so that the joint assumes undue mobility, and spontaneous luxations occur. Spontaneous fractures in connection with supposed symptoms of paralysis have, from a remote period, been observed by surgeons; but it is probable that the symptoms were really ataxic and not paralytic. It is very probable that these arthropathies are dependent, as Charcot asserts, upon disease of a certain group of the large ganglion cells of the anterior horns. In two cases examined by Charcot and Joffroy the anterior horns of grey matter were found remarkably atrophied, and a considerable number of the large ganglion cells, especially of the postero-lateral group, were atrophied or completely destroyed. In unilateral articular affections of this kind these changes were observed only on the corresponding side of the cord, and when the shoulder joint was exclusively affected the disease was found only in the cervical region, and when the knee joint was alone implicated the disease was found limited to the lumbar portion of the cord. In three cases examined by Joffroy, and in one observed by Coyne, no alteration of the cells of the anterior horns was found.

*Fractures.*—Spontaneous fractures have attracted the attention of surgeons from a remote period, but these accidents were attributed to the influence of certain diatheses, such as gout, rheumatism, scrofula, and cancer. Larrey drew special attention to the fact that a certain form of paralysis of the lower extremities was associated with a strong predisposition to fractures of



their bones. In the record of this case, however, it is mentioned that the so-called paralytic symptoms were associated with amaurosis and great exaltation of the sensibility of the lower extremities, which renders it almost certain that the symptoms were not due to paralysis, but to ataxia. In 1873 Weir Mitchell drew attention to the frequency of spontaneous fractures in locomotor ataxia, and suggested that during the progress of the disease the bones had undergone nutritive changes which greatly diminished their resistance. This subject was subsequently investigated by Charcot and his followers, with their usual thoroughness and success. The period of fracture is generally preceded by two or three paroxysms of lancinating pain of unusual severity, and at the same time the limb is found swollen, and with all the symptoms of osteo-periostitis; and fracture occurs on the slightest movement of the limb, or in the entire absence of any movement or other external cause. The femur is more frequently fractured than any other bone, the seat of fracture being frequently the neck of the former; but the bones of the leg, arm, forearm, and indeed almost every bone of the limbs and trunk, have been found fractured, including the vertebral column. Multiple fractures in the same patient are by no means uncommon, and in a case published by Charcot, the patient, towards the close of life, could scarcely move in bed without fracturing some one or other of the few bones which had not already been fractured. Damaschino has drawn attention to the fact that the spontaneous fractures of ataxics reunite very readily and rapidly with an enormous formation of callus.

§ 120. *Osseous Affections of Cerebral Origin.*—The arthropathies of hemiplegic patients, which were first described by Scott Alison, Brown-Séquard, and Charcot, are of the same kind as the acute arthropathies of spinal origin. Cerebral arthropathies have been observed in connection with softening, hæmorrhage, and intracranial tumours, but they are more frequently associated with softening than either of the other lesions. This articular affection usually attacks the joints of the hand or foot, and only very rarely the elbow or knee joint. The affection begins with a slight swelling and local increase of temperature,

either with or without pain in the joint; and at times the tumefaction and redness are so marked as to resemble the articular affections of acute rheumatism. The sheaths of the tendons are sometimes affected along with the joints. These arthropathies occur usually at the time late rigidity sets in, or from fifteen days to a month after the attack of apoplexy. They may, however, appear at a much later period; while Weir Mitchell, on the other hand, observed in one case arthritis three days after an attack of apoplexy, and in a second case the joint affection showed itself the day following the cerebral attack. The nature of the affection was shown by Charcot to be a true synovitis with multiplication of the nuclear and fibroid elements of the articular serous membrane. In severe cases a sero-fibrinous fluid mixed with white blood corpuscles is exuded, which may be sufficiently abundant to distend the synovial cavity. The tendinous synovial sheaths in the neighbourhood of the affected joints participate in the inflammation.

The frequent coincidence in time of the appearance of these arthropathies and of late rigidity would appear to indicate that the former are due to descending changes in the pyramidal tract and probably also secondary implication of the ganglion cells of the anterior cornua of the spinal cord; but in two cases observed by Charcot the autopsies showed complete absence of every change in the nerves and spinal cord, and of secondary descending sclerosis of the lateral columns. Hitzig thinks that the arthritis is of traumatic origin and due to the displacement of the surfaces of the joints, caused by paralysis of the muscles surrounding them. This explanation may suffice for the arthropathies which occur long after the paralysis has become established; but it will not account for the joint affections occurring a few days after the attack. Scott Alison attributed the arthritis to diminution of the vitality of the affected parts, permitting the previously existing poison of the uric acid diathesis to act on the joints of the paralysed limbs. Charcot also appears to incline towards this theory, and adduces in its favour the observation that, at an autopsy of a hemiplegic patient, in the joints, which during life had frequently been the seat of pain and swelling, deposits of urate of soda were found, while nothing of the kind was observed in the joints of the opposite side.



§ 121. *Osseous Lesions in the Insane.*—Attention has of recent years been directed, especially in England, to the very important morbid changes of the osseous system which occur amongst the insane. These affections are of two kinds, although they are probably fundamentally the same. In one of these the bones become so soft that they yield readily to pressure and thus produce various deformities. In the other form the bones become so fragile that they are found after death to crumble under the finger and thumb, and are, of course, during life liable to fracture. The morbid changes which occur in the bones of the insane are closely related to those which occur in the spontaneous fractures of locomotor ataxia.

(V.)—NUTRITIVE AND SECRETORY AFFECTIONS OF THE  
GLANDULAR APPARATUS.

It was shown by Pflüger that the nerves of the submaxillary gland terminate in fine threads, which are connected with and terminate in the secretory cells of the gland, and it is very probable that the secretory nerves of other glandular organs have a similar termination. With respect to the lachrymal gland, Boll has observed fine terminal threads distributed between the cells of the gland. The various experimental and pathological facts which have been collected show that the secretory nerves are quite independent of the vaso-motor nerves of these organs, and that not merely the secretion but even the development of the glands is under the influence of these nerves (Heidenhain).

§ 122. *Trophoneuroses of the Salivary Glands.*—Secretory disturbances of the large salivary glands may occur in connection with lesions of the peripheral fibres of the trigeminus, of the facial nerve, or of the cervical sympathetic. In trigeminal neuralgia an increased secretion of saliva is not an unfrequent symptom, due to reflex irritation conveyed through the lingual nerve of the fifth as the afferent, and the chorda tympani as the efferent channel. Stimulation of the glosso-pharyngeal is even more effectual in increasing the flow of saliva than stimulation of the lingual nerve. If the chorda tympani be divided the flow of saliva from the submaxillary gland is arrested from want

of efferent impulses; but if the peripheral portion of the nerve be stimulated a copious secretion of a thin watery saliva at once takes place, while the arteries of the gland become dilated. That the increased secretion does not depend upon the vaso-motor action of the stimulated nerve is shown by the fact that when the chorda tympani is energetically stimulated the pressure acquired by the saliva in the duct exceeds for the time the arterial blood-pressure.

In peripheral paralysis of the facial nerve there is often a diminution of secretion of saliva on the paralysed side. This is due to the fact that the chorda tympani contains the secretory fibres for both the submaxillary and sublingual glands; while the secretory fibres of the parotid are contained in the small superficial petrosal nerve. In paralysis of the cervical sympathetic a diminution of salivary secretion may be observed on the affected side, arising from the fact that the parotid obtains a portion of its secretory fibres through the cervical sympathetic. Certain poisons, as atropine, paralyse the secretory fibres of the chorda tympani, and consequently lead to a diminution or arrest of the salivary secretion; other substances, as digitalin, physostigmin, nicotin, and, before all, jaborandi, appear to increase the action of the salivary secretory fibres and lead to an increased flow of saliva.

The secretion of saliva may be influenced by direct or reflex action on the intracerebral secretory paths. Bernard found that an increased flow of saliva might be produced by puncture of the floor of the fourth ventricle behind the origin of the trigeminus. It is possible that the increased flow of saliva observed in bulbar paralysis may at times be due to irritation of this point. An enormously increased flow of saliva has been observed by Eulenburg in dogs after destruction by the actual cautery of portions of the cortex of the brain lying in front of the cruciate sulcus. The saliva flowed out in a constant stream from the angle of the mouth on the opposite side to the injured hemisphere, and was of the same thin watery character which is observed after irritation of the chorda tympani.

§ 123. *Trophoneuroses of the Lachrymal Glands.*—Increased lachrymal secretion, like increased salivary secretion, often occurs



in trigeminal neuralgia, more especially of the two first divisions. It is caused partly by direct and partly by reflex irritation of the secretory nerves, which, according to the experiments of Herzenstein and Wolferz, are partly contained in the lachrymal nerve and partly in the subcutaneous malar nerve. Electrical irritation of these nerves caused increased secretion of tears on the side of the irritation; and the same result can be induced in a reflex manner by peripheral irritation of the first and second divisions of the trigeminus. Irritation of the branches of the trigeminus may also give rise to an increased secretion of a watery, mucous, or even bloody fluid from the mucous membrane of the nose, which may also be caused by irritation of the sphenopalatine ganglion, and of the nerve branches which spring from it.

The diminished flow of tears and of the secretion of the mucous membrane of the nose, not unfrequently observed on the affected side, in unilateral facial atrophy is probably due to an affection of the ganglion of the trigeminus, or of individual branches of the trigeminus. Affections of the sympathetic may also cause disturbance of the lachrymal secretion. After traumatic injury of the cervical sympathetic, increased flow of tears is often observed on the affected side, which is usually accompanied by redness and a rise of temperature on the same side of the head, and by congestion of the conjuction. These phenomena are probably due to vaso-motor paralysis, and the copious flow of tears, which often takes place at the end of an attack of hemicrania, is probably of a similar character. Many anomalies in the flow of the lachrymal secretion are no doubt of central origin, such as the copious flow which occurs in hysterical attacks, and in connection with emotional disturbances, and these are often associated with similar anomalies of other secretions. Parrot, for instance, has observed a bloody condition of the tears in connection with a similar condition of the perspiration during severe hysterical convulsions.

§ 124. *Trophoneuroses of the Glands of the Digestive Tract.*—The probable dependence of diabetes mellitus upon the vaso-motor innervation of the liver has already been mentioned. Very little is known with respect to the action of the nervous system on the formation of bile. Pathological increase of the secretions of the stomach and intestines appears at times to depend partly on affections of the vagus, and partly on affections of the sympathetic plexuses and ganglia. These anomalies often depend upon a central disturbance, as, for instance, the

vomiting and diarrhoea which are not unfrequently caused by emotional disturbances. The considerable increase of the secretion of the stomach observed in cases of hysterical vomiting is probably due to irritation of the vagus, either in its course or in its central origin. The vomiting of hemicrania, and the increased secretion observed in cardialgia and pyrosis, are also due to vagus irritation, either at its peripheral or central origin. Considerable diminution of the secretions of the stomach and intestines, giving rise to imperfect digestion, wasting, and habitual constipation, is a frequent accompaniment of grave neuroses like hysteria, hypochondriasis, and mental diseases; or may be induced by various toxic agents, such as opium and the preparations of lead.

§ 125. *Trophoneuroses of the Glands of the Genito-Urinary Apparatus.*—The influence of the nervous system on the secretion of urine is very great, but the various channels by means of which it is conveyed are not accurately ascertained. Some of the pharmaceutical agents which increase the secretion appear to produce a direct local irritation of the secreting nerves of the kidney, while other agents increase the secretion by raising the arterial tension. Various anomalies of secretion may occur from the uterus, vagina, and mammæ in hysterical females, although it is doubtful in these cases whether the disturbance is due to affections of the vaso-motor or of special trophic nerves. In the condition which has been described under the name of irritable uterus, hysteralgia, and other terms, there are often, in addition to the sensory disturbance, numerous anomalies of circulation and secretion, which are probably of reflex origin. Similar phenomena may be associated with cutaneous neuralgia, and more especially with ilio-lumbar neuralgia. In hysterical patients directly after a paroxysm there is frequently observed an abundant secretion of mucus, or an increase of an already existing discharge. Erotic thoughts may give rise to an obstinate mucous discharge from the vagina in the absence of any organic lesion. Most of the pharmaceutical agents which increase or diminish the secretion of milk appear to act through the nervous system.

In men the condition known as "irritable testis" is often



like the "irritable uterus" associated with anomalies of circulation and nutrition. Swelling of the testicle and of the spermatic cord, dilatation of vessels, and varicocele are often observed along with neuralgia of the testis; and these are sometimes of a primary, sometimes of a secondary nature. Many cases of spermatorrhœa, pollutions, and aspermatism may probably be attributed to functional disturbances of secretory or motor nerve fibres arising in the lumbar portion of the spinal cord. Pollutions appear generally to depend upon an increased reflex irritability from peripheral irritation, or upon increased excitability of the centre of ejaculation in the spinal cord. The latter condition often occurs in grave diseases of the cord, such as *tabes dorsalis*; and it is sometimes associated with alterations in the quality of the semen, as immobility or even complete absence of spermatozoa. Spermatorrhœa, on the other hand, which is not of purely mechanical origin, as that caused by enlargement of the prostate gland, depends less upon an increase of the secretion than upon a debility of the *vesiculæ seminales*, or of the spermatic conduits.

(VI.) NUTRITIVE AFFECTIONS OF THE VISCERA (VISCERAL TROPHONEUROSES).

§ 126. It has not yet been found possible to separate the vaso-motor and trophic fibres of the viscera with respect either to their anatomical distribution or their functional capacities; and, indeed, the existence of the trophic fibre can scarcely be said to have been proved. It is therefore very doubtful whether to regard any particular visceral neuroses as a vaso-motor or trophic affection. It is more than probable that the congestion, ecchymoses, and extravasations, and probably also various forms of hydruria and albuminuria which occur in connection with various central nervous diseases, are due to implication of the vaso-motor nerves.

Notwithstanding numerous experiments on animals, it is still doubtful whether the thoracic and abdominal ganglia and plexuses contain, besides the vaso-motor fibres, specific trophic fibres for the viscera; and whether, as was first asserted by Axmann, these fibres arise out of the spinal ganglia. The asser-

tions of numerous experimentalists, amongst whom may be mentioned Bernard, Samuel, Budge, Adrian, Schmidt, and Schiff, are extremely contradictory. The most frequent consequences of extirpation or destruction of the cœliac and mesenteric plexuses are congestion and extravasation of the stomach and intestines, enlargement and congestion of the liver, and diabetes, all of them symptoms which are probably due to vaso-motor paralysis. The reaction of the operation on the organism as a whole is also very variable, some authors having noted a transitory emaciation, while others found that animals have become fatter after extirpation of the cœliac ganglion.



## CHAPTER VII.

## GENERAL MORBID ANATOMY AND PHYSIOLOGY.

It will be useful to consider the leading outlines of the morbid anatomy of the nervous system, before entering upon details in the special part. The nervous tissues consist, as already mentioned, of cells and fibres packed together by means of connective tissue (neuroglia) so as to form organs, and supplied with blood-vessels, lymph spaces and vessels, and with blood. Now the nerve cells and fibres may themselves be primarily diseased, constituting the *parenchymatous affections* of the nervous system; or they may be secondarily diseased, the primary affection occurring in the connective tissue, the vessels, or the blood; or the disease may begin in neighbouring organs and extend from these to the nervous tissues. But inasmuch as the parenchymatous diseases always cause secondary changes in the connective tissues, blood-vessels, and blood, and the diseases of the latter react on the parenchyma, it is not by any means always easy to distinguish between primary parenchymatous disease on the one hand, and the diseases of the connective tissues, along with the vascular and toxic neuroses, on the other. The term *lesion* is used as a generic expression for any morbid alteration of tissue, whether this alteration be or be not attended by such structural changes as can be recognised after death by our means of research.

§ 127. *Classification of Lesions of the Nervous System.*

Lesions of the nervous system may be classified: I. According to their nature; II. According to their form; III. According to the functional disturbances they produce.

(I.)—CLASSIFICATION ACCORDING TO THE NATURE OF THE LESION.

(1) *Inflammation*.—Every part of the nervous system is subject to inflammation, which, like inflammation of other tissues, may be acute, sub-acute, or chronic with respect to its course and development. When the affection is acute it generally ends in complete disintegration of the affected tissue, which, on being mixed up with fluid and morphological elements effused from the blood-vessels, presents a pulpy mass technically called *softening*. When the inflammatory process is chronic the tissue undergoes degeneration; but inasmuch as degeneration may occur independently of inflammation, both kinds may be included in a separate group.

(2) *Degenerations*.—Degenerations of nervous tissues are of various kinds; but inasmuch as in all of them the nervous tissue, instead of undergoing softening as in acute inflammation, becomes somewhat denser than usual, they are called *scleroses*. From the colour of the altered tissue, it is sometimes called *grey degeneration*. Both inflammations and degenerations include affections which have begun in the connective tissues or neuroglia, the vessels and their adventitiæ, or the blood itself, as well as those which are primarily of parenchymatous origin.

(3) *Vascular Lesions*.—Besides the vascular lesions which accompany all inflammatory and degenerative processes, other very important diseases in connection with the vessels must be mentioned.

(i.) *Hyperæmia and Anæmia*.—The vessels are at times actively dilated so that an undue quantity of blood is sent to portions of the nervous system, while they are at other times contracted so that the normal quantity is diminished. The hyperæmia may at times be active and due to high arterial tension and active dilatation of the arterioles, while at other times it is passive, and is then caused by some obstruction of the blood along the large veins of the body. The anæmia to which the nervous system is subject do not differ from the anæmia of other organs, and may therefore be due to an alteration of the quality as well as the quantity of the blood.

(ii.) *Hæmorrhage*.—Rupture of vessels with consequent hæmorrhage into an organ is a very common cause of disease



of the central nervous organs, and especially of the cerebrum. The rupture may at times be due to accidental injury, but more frequently it occurs in the degenerative period of life, and is then caused by various degenerations of the coats of the vessels, such as atheroma and the fibrosis which accompanies Bright's disease.

(iii.) *Embolism and Thrombosis*.—The morbid processes of embolism and thrombosis, when they occur in the nervous system, are essentially the same as in any of the other organs of the body. The sudden arrest of the circulation, caused by obliteration of an artery, causes intense anæmia and loss of function of the part to which its branches are distributed. The centre of the ischæmic region usually undergoes necrobiosis, and its substance becomes altered into a soft pulpy mass closely resembling inflammatory softening.

(4) *Toxic Lesions*.—Various chemical agents circulating in the blood induce morbid alterations of the nervous tissues.

(5) *Traumatic Injuries*.—Wounds, contusions, and other traumatic influences cause so many alterations of nervous tissues that their results deserve to be mentioned amongst the morbid lesions of the nervous system.

(6) *Compression of Nervous Tissues*.—The nervous tissues are frequently subjected to sudden or gradual compression from various causes. One of the most frequent causes of compression is the gradual encroachment on the tissues of new formations, no matter whether the growth be outside the nervous tissues or *extra-neural*, or in the substance of the tissues or *intra-neural*. Other causes of compression are fractures of the cranium, dislocations, fractures and curvatures of the vertebræ, the formation and subsequent enlargement of abscesses and aneurisms, and the growth of cysticeri and other parasites. Inflammatory effusions and thickenings of the membranes of the brain or cord, or of the sheaths of nerves, also injure the nerve tissues by compression as well as by extension of the morbid process to the nervous tissues themselves.

## (II).—CLASSIFICATION ACCORDING TO THE FORM OF THE LESION.

(1) *Circumscribed or Focal and Diffused Lesions—Systematic Diseases*.—When a lesion is circumscribed within

definite limits it is called a *focal lesion*, and when it extends over an indefinite area with irregular limits it is called a *diffused lesion*. When the lesion is limited to a portion of the nervous system which possesses a distinct functional unity, it is called a *system-disease* or a *systematic lesion*. A lesion limited to the pyramidal tract in the spinal cord is a good example of a system-disease.

(2) *Molecular, Molar, and Histological Lesions*.—In order to study these diseases under the simplest conditions, let us suppose that the sciatic nerve of a frog is isolated, with the gastrocnemius muscle attached. On being stimulated by a strong faradic current, the muscle immediately contracts; but a second shock through the nerve is powerless to induce a contraction—the nerve is paralysed. During the passage of the nerve from almost perfect health to complete, though temporary, paralysis, the change which it has undergone is such as cannot be detected by the most refined chemistry, or by the aid of the highest powers of the microscope. In such a case we assume that the molecules of the axis-cylinder have come to a condition of stable equilibrium; hence the cause of the loss of function may be described as a *molecular lesion*. It need scarcely be added that a molecular lesion may also give rise to excess, as well as diminution, of functional activity. The lesion which I have called *molecular* has also been termed *functional*, from the fact that the part affected, while giving rise to definite functional deviations, does not present any apparent structural changes.

But if a portion of the sciatic nerve be crushed or cut, the conductivity of the nerve is destroyed at the point of injury, and a faradic current applied to the nerve on the central side of the injury will not cause the muscle to contract. The loss of function in this case is caused by a lesion, which can be recognised by the naked eye; hence it may be called a *molar lesion*. Dr. Hughlings Jackson has proposed to call the molar lesion by the name of “coarse disease,” and the molecular lesion by the name of “fine disease”—names which, at least, possess the merit of explaining themselves. Between the fine or the molecular lesion on the one hand, and the coarse or molar lesion on the other, another variety may be interposed. When the



morphological elements of the nervous tissues themselves, or of the tissues by which the nervous elements are surrounded, undergo alterations which can be recognised by the aid of the microscope, the morbid change of structure may be called a *histological lesion*.

(III).—CLASSIFICATION ACCORDING TO THE ALTERATIONS OF FUNCTION PRODUCED BY THE LESION.

(1) *Irritative and Depressive Lesions*.—When the morbid alteration is attended during life with excess of functional activity, it is inferred that the lesion is one of an *irritative character*, or, in other words, it is inferred that the irritability of the cells and fibres of the part affected is increased. The opposite condition, when the irritability is diminished or abolished, deserves a special name, and may be called a *depressive lesion*.

(2) *Discharging and Destroying Lesions*.—The morbid alterations which are attended by paroxysmal and excessive liberations of energy have been called by Dr. Hughlings Jackson *discharging lesions*. We have seen that the nerve cells are the main generators and accumulators of energy, hence these lesions always implicate the grey substance, although it is not always easy to draw a sharp line of distinction between discharges of energy from grey substance and those which result from irritation of nerve fibres. When the affection is accompanied by a distinct destruction of nerve tissue, such as occurs in hæmorrhage into the substance of the brain, Dr. Hughlings Jackson has named it a *destroying lesion*.

§ 128. *Relation between Morbid Nervous Structures and Functions*.—It has already been stated that when the irritability of the nervous tissues is increased, or, in other words, when the molecules of the active portion of these tissues occupy positions of unstable equilibrium, the functional activity of the part affected is increased; and, conversely, when the molecules occupy relatively stable positions, the functional activity of the part is diminished. The causes which raise or depress the irritability have already been described (§ 16), and when one of these causes is operative the capacity of the affected part for

function undergoes corresponding variations. Increased irritability of a part demands increased activity of material exchanges, which in its turn renders necessary an increased supply of nutriment; and, conversely, when the irritability is depressed the material exchanges and the nutrient supply are diminished. It may be inferred, therefore, that a free arterial supply to a part, or a flushed condition of the arterioles, is the necessary correlative of increased irritability; and, conversely, that a diminished arterial supply, with an empty and contracted condition of the arterioles, is the necessary correlative of diminished irritability. These statements, however, can only be accepted as true within certain limits and with numerous qualifications. When the brain, for instance, is very freely supplied with blood so that its substance becomes congested, the irritability of the tissues is no doubt at first increased. It must, however, be remembered that the cranium is unyielding, and its contents practically incompressible, so that no additional quantity of blood can enter into the intracranial vessels except by displacing a corresponding quantity of some other fluid. When, therefore, the vessels become dilated beyond certain narrow limits the nervous tissue becomes compressed, the material exchanges within the cranium become less than when the circulation passes in normal quantity and under normal pressure, and the functional activity of the organ is diminished or abolished. A similar process no doubt occurs in the spinal cord and nerve trunks. Congestion in them, when carried beyond certain limits, is also attended with diminution of function, due, no doubt, to compression of the nerve tissues by the dilated vessels. The *irritative lesion* is attended with increased nutritive activity, and consequently with free arterial supply; but this lesion is exceedingly apt to terminate in the opposite condition of diminished nutrition and functional activity. The first stage of inflammation, for instance, is an *irritative* lesion, and it is attended by excess of functional activity, manifesting itself by symptoms of hyperæsthesia and hyperkinesis; but when the nervous tissues become partly compressed by effused products and partly disorganised by internal changes, the lesion becomes *depressive*, and the symptoms of excess give place to those of diminution of func-



tion; in other words, the symptoms of hyperæsthesia and hyperkinesis give place to those of anæsthesia and akinesis.

But if excess of nutrient activity is not always accompanied by increased functional activity, neither is diminished nutrient activity always accompanied by diminution of functional activity. We have indeed seen (§ 16) that when the nutrition of a nerve fibre is gradually lessened its irritability is increased instead of being diminished. The stock of irritable matter which the nerve fibre possesses is no doubt less under these circumstances, but an increased readiness to discharge the energy is manifested; and it is notorious that feeble and anæmic persons manifest an undue readiness to respond to the action of stimuli of all kinds, a condition which is correctly designated *nervous irritability*. One other important consideration must be taken into account before the amount of nourishment supplied to an organ or a tissue can be accepted as in any way a measure of the functional activity of the latter. We have seen that, when a strong faradic current is sent through the sciatic nerve of a frog, the gastrocnemius muscle contracts strongly; but a subsequent current passed through the nerve is followed by no reaction until the irritability of the nerve is restored by the absorption of more nourishment. A similar process no doubt occurs in disease of the nervous system. When a part is supplied with an excessive amount of nourishment, the tissues become so irritable that they discharge readily, either spontaneously, or in response to stimuli which would not affect them under normal conditions. Under these circumstances, excessive discharges of nervous energy readily take place, and these are followed by temporary loss of irritability, and the tissue becomes incapable for a time of performing its normal functions. The excessive liberations of energy from the cortex of the brain, producing epileptic attacks, for instance, are accompanied by loss of consciousness, which lasts for a considerable time, and the convulsive phenomena are not unfrequently followed by temporary motor paralysis. The energy of the *discharging lesion* being liberated, the part affected is rendered incapable of performing its functions, until its irritability is restored by the absorption of a fresh stock of irritable matter. The primary effect of almost all chemical

agents on the nervous system is to stimulate it and to increase its functional activity, while their secondary effect is to depress or abolish its functional activity. The stimulant action of alcohol on the brain, for instance, is followed by a stage of depression, which may amount to complete abolition of the cerebral functions or coma. Strychnine increases the irritability of the grey substance of the spinal cord; but the reflex actions, which are at first greatly exaggerated, become ultimately abolished, and the animal poisoned by strychnine often dies from paralysis. Curara, which may be taken as the type of nervous sedatives, paralyses the terminations of the motor nerves, yet Bernard proved that it first increases the irritability of the fibres.

The direct tendency of all destroying lesions is to abolish function. It must, however, be remembered that these lesions are frequently surrounded by a zone of nervous tissue, which is in a state of irritation, and the prominent symptoms of the affection are often produced by this zone, consequently the symptoms are indicative of excess of functional activity. A gummatous tumour, for instance, in the cortex of the brain is generally declared by epileptoid convulsions; yet the direct tendency of the tumour, in so far as it has destroyed and replaced nervous tissue, is to abolish function. In such cases, both the direct and indirect effects of the tumour are often manifested; the former by paralytic and the latter by convulsive symptoms. Even the ischæmic softening, caused by plugging of vessels, is often surrounded by a congestive zone of tissue, and the latter may give rise to symptoms of irritation.



## CHAPTER VIII.

## GENERAL DIAGNOSIS AND PROGNOSIS.

## (I).—DIAGNOSIS.

THE diagnosis of disease of the nervous system may be divided into (1) the Topographical Diagnosis and (2) the Pathological Diagnosis.

§ 129. *Topographical Diagnosis*.—The aim of topographical diagnosis is to determine with accuracy the *seat* of the affection, or the *localisation* of the lesion, as it is now called. This subject has been incidentally treated of when describing and, to a certain extent, interpreting the symptoms of nervous disease; hence only brief reference will be made to it at present. One useful rule in determining the localisation of the disease is to place the *seat* of the lesion at a point where all the affected paths lie near together. This rule, however, demands considerable caution in its application, inasmuch as a plurality of lesions is frequently observed. The most practically useful classification of nervous diseases for the purposes of topographical diagnosis is that which divides them into (1) Peripheral, (2) Spinal, and (3) Encephalic lesions.

(1) *Peripheral Lesions*.—In peripheral lesions the functional disorders are generally limited to single nerves or branches of nerves, and the location of the sensory, motor, vaso-motor, and trophic disturbances coincides with the distribution of the affected nerve. The presence of muscular atrophy, especially when the muscles are supplied by a single nerve, combined with the appearance of the reaction of degeneration in the affected muscles, the complete absence of all reflex action, and of all weakness of the bladder and of the

sexual functions, are in favour of peripheral paralysis. Acute disease of the anterior grey horns of the cord gives rise to muscular atrophy, the reaction of degeneration, absence of the reflexes in the paralysed muscles, while the bladder and sexual functions are unaffected; but in the latter case the muscles affected are grouped according as they are associated in their actions, while in peripheral paralysis they are grouped according to the distribution of individual nerves and their branches. Valuable information may be obtained from the history of the onset of the affection, which will enable us to distinguish between peripheric paralysis and atrophic spinal paralysis. The latter disease occurs suddenly, and its onset is generally attended with severe general symptoms, and it occurs more frequently in childhood than in later life; while the former disease may occur at any period of life, and when it occurs suddenly it is generally caused by some injury in the course of the nerve, which leaves a permanent cicatrix or other indication of the peripheral origin of the lesion. In peripheral paralysis, anæsthesia, limited to the distribution of the affected nerve, is generally present; while the spinal forms of atrophic paralysis are characterised by complete absence of sensory disturbances.

It has been asserted by Weir Mitchell that, in disease of the peripheral nerves, retardation in the conduction of sensation is never manifested. When a tumour simultaneously presses upon a large number of nerves, such as the cauda equina, or on the nerves at the base of the skull, it is not always easy to distinguish between paralysis of peripheral and of central origin. The rules to be followed in distinguishing those affections must be reserved for the special part.

(2) *Spinal Lesions*.—The most characteristic feature of spinal diseases is a nearly uniform ascending paralysis of both sides, constituting *paraplegia*. The symmetrical groups of muscles of the lower extremities, trunk, abdomen, and upper extremities are paralysed progressively from below upwards, according as the disease ascends from the lumbar through the dorsal to the cervical region of the cord. Sensory disturbances are also usually present, though they are not always equal in extent and intensity to the motor affections. When the motor paralysis is limited to one half of the lower part of the body (*hemi-paraplegia*) the



sensory and motor disturbances cross, so that the one occupies the one side and the other the corresponding parts on the opposite side. The spinal sensory disturbances frequently consist merely of retardation of sensory conduction and various paræsthesiæ, such as numbness, formication, and girdle sensations at the upper limit of the motor affections. Sensory disturbances are absent in the spinal paralysis of children and allied forms, a fact which constitutes an important element in the diagnosis between them and atrophic paralysis of peripheral origin. Spinal affections are also frequently accompanied by disturbances in the functions of the bladder, rectum, and sexual organs, and where the cervical region is affected paralytic myosis is often present. The condition of the various reflexes, both superficial and deep, are exceedingly valuable in the diagnosis of spinal affections. These reflexes may be either exaggerated, diminished, or abolished, and thus valuable information is afforded, both with regard to the nature of the lesion, whether irritative or depressive, and to the level at which the cord is affected. The various vaso-motor and trophic affections, especially the appearance of acute bed-sores over the sacrum, also afford valuable evidence of the presence of spinal disease; while the absence of disturbances of the cranial nerves, including the special senses, and of all psychical disturbances, aid us in distinguishing spinal from cerebral disease.

(3) *Encephalic Lesions.*—The main points on which we rely in distinguishing cerebral from spinal and peripheral disease are the hemiplegic distribution of the sensory and motor disturbances, both being limited to the same side of the body, and generally of very unequal intensity; the absence of all trophic disturbances, normal electric reaction of muscles and nerves, and retention or exaggeration of all reflex acts, and the retention of associated and automatic movements, and of the rectal and vesical functions. In intracranial disease the cranial nerves are frequently implicated, while disturbances of speech and of the higher mental faculties, both intellectual and emotional, are generally observed. The presence of headache, giddiness, and unaccountable vomiting also afford valuable diagnostic signs of many forms of cerebral disease. The diagnostic significance of the various forms of spasm and convulsion,

muscular tension, contractures, and the different forms of tremor, is too complicated a subject to be fully discussed at present, and must be therefore reserved for future mention.

§ 130. *Pathological Diagnosis.*—The aim of pathological diagnosis is to determine the *nature* of the lesion, no matter where it may be situated. It may be laid down as a general rule, but one requiring numerous qualifications and cautions in practice, that a lesion which, in the absence of traumatic and toxic influences, develops symptoms of depression in the course of a few minutes, or a few hours, is of *vascular* origin; that a lesion which takes from a few days to a few weeks for the full development of the symptoms, and in which the symptoms of irritation are followed by those of depression, is of *inflammatory* origin; and that a lesion which takes from two to six months, or longer, for the full development of its symptoms, and in which the primary symptoms of irritation are either absent or obscured by the more prominent symptoms of depression, is of *degenerative* origin, or due to a slow and gradual compression of nervous tissue by the growth of a new formation.

(1) *Vascular Lesions.*—The vascular lesions, which are the most frequent causes of disease of the nervous system, are rupture and hæmorrhage, embolism, and thrombosis. The lesion is more likely to be due to hæmorrhage if the affection occur after forty-five years of age, and during the degenerative period of life, if the arteries at the wrist and temples be hard and knotty, if there be some hypertrophy of the left ventricle along with an accentuated second sound of metallic quality in the aortic area, if the patient be suffering from chronic Bright's disease, and its attendant degeneration of arteries and high arterial tension, and if a well marked arcus senilis or other signs of degeneration of tissue be present. It is also probable that emphysema and cardiac diseases favour hæmorrhage by producing passive congestion of the brain.

The lesion is more likely to be embolic if the affection occurs prior to the degenerative period of life, if there is valvular disease of the heart; and, where the affection is of cerebral origin as it generally is, if loss of speech or aphasia be a prominent symptom. When the lesion is due to thrombosis the symptoms



are as a rule slower in their development than in hæmorrhage or embolism. The signs which favour the idea that the lesion is due to thrombosis are, the supervention of symptoms more or less similar to those which would be caused by hæmorrhage or embolus, along with evidence that the arteries are affected either by atheroma, calcification, or syphilitic endo arteritis.

(2) *Inflammatory Lesions.*—The inflammatory nature of the lesion must be determined by the mode of onset being more gradual than in vascular lesions, the symptoms of depression being preceded by those of irritation, and the presence during the development of the affection of elevation of temperature and other febrile symptoms.

(3) *Degenerative Lesions.*—In the degenerative lesions the mode of onset is still more gradual than in the inflammatory lesions. Symptoms of irritation are almost or entirely absent during the whole progress of the case; while there is entire absence of pyrexia as the result of the lesion.

(4) *New Formations.*—Tumours which compress the peripheral nerves can usually be detected by ordinary physical examination, and it is only in the case where the tumour occupies the vertebral canal or the cavity of the cranium that any special difficulty of diagnosis presents itself. The development of the symptoms produced by the growth of tumours is never acute, and rarely very chronic, but no great reliance can be placed on this fact as evidence in diagnosis. The most valuable evidence is, indeed, obtained from the history of the development and the grouping of the symptoms; and the presence of an intra-vertebral or intracranial tumour must be inferred principally by the method of exclusion; or, in other words, by showing that the symptoms could not be caused by any other known disease. After having determined that the symptoms are due to the growth of a new formation, the question as to the *nature* of the growth remains unanswered. It would lead us too far to enter on this wide subject at present; but much valuable information with reference to this question may be obtained from a close study of the constitution of the patient, taking special account of hereditary influences. If, for instance, the symptoms of intracranial tumour are present in a child, whose sister has suffered from scrofulous swelling of the knee-

joint, or in whose family history other unmistakable evidence of scrofula can be discovered, it is probable that the tumour will be of scrofulous origin. It is impossible at present to enter upon the wide subject of the diagnosis of syphilitic affections of the nervous system further than to warn the student of the extreme importance of the subject; for inasmuch as there is no severe organic disease affecting the nervous system in which the results of treatment are so often satisfactory, so there is no disease which deservedly brings so much discredit upon the practitioner who overlooks its presence.

The causes of disease, both exciting and predisposing, afford valuable aid in diagnosis. A history of the patient having been exposed to excessive fatigue, cold, or traumatic influences, or having recently suffered from an attack of one of the acute specific fevers, including diphtheria, or evidence which would lead to the suspicion of some poison having been taken, may afford valuable information to aid us in forming a correct diagnosis.

#### (II.)—PROGNOSIS.

§ 131. It may be stated as a general law that, other things being equal, the prognosis is more favourable in affections of the sensory than of the motor mechanism. We have already seen that the efferent impulses pass through much more definite channels than the afferent impulses. The voluntary efferent impulses issue from large caudate cells in the cortex of the brain, pass downwards through distinct medullated nerve fibres, to reach the large ganglion cells of the anterior grey horns of the spinal cord, the latter being connected by distinct axis-cylinder processes with the efferent fibres of the peripheral nerves. The structure of the sensory presents a striking contrast to that of the motor mechanism. It is true that afferent currents from the periphery are conveyed by medullated fibres to the spinal cord through the posterior roots; but these fibres are smaller in diameter than the corresponding fibres of the anterior roots, and on reaching the grey substance they do not appear to form any definite connections with the cells of the posterior horns, nor do they appear to be continued upwards uninterruptedly through the cord to the cortex of the brain. It seems indeed as if the sensory conduction through



the cord were largely effected by the grey substance, and in a diffused manner. And when the afferent fibres of the corona radiata reach the cortex of the brain, instead of terminating by definite connections with the caudate cells of the internal layers of the cortex, they are continued upwards to the small cells of the superficial layers, amongst which they terminate, but without forming any definite connections with them. The definite structure of the motor mechanism would, therefore, lead us to expect that when any of its fibres and cells are destroyed a more or less enduring loss of conduction and paralysis will result; while the indefinite structure of the sensory mechanism would, on the other hand, lead us to expect that when some of its cells and fibres are destroyed the afferent impulses, temporarily arrested, will readily find new channels of conduction in the neighbouring cells and fibres. This expectation is realised in practice. Disease or injury of the motor mechanism is known to produce much more definite and enduring symptoms than disease of the sensory mechanism.

In the spinal cord in animals, for instance, experimental section of the lateral column produces complete paralysis of the posterior extremity of the same side; while complete loss of sensation of the posterior extremities does not result so long as a small portion of the grey substance of the cord is left intact. It is evident, therefore, that sensory conduction is much more diffused than motor conduction. Even in peripheral paralysis of traumatic origin, when regeneration of the nerve takes place, sensibility reappears in the affected parts at a much earlier period than motor power. This fact has led Baerwinkel to propose a valuable prognostic test in cases of severe traumatic paralysis. If in the first few months after injury pressure on the nerve below the seat of the lesion is followed by an excentric sensation, it may be expected that regeneration will take place, and that motor power will subsequently return.

It may also be laid down as a general law, requiring numerous qualifications, that, other things being equal, peripheral are less serious than central affections, and cerebral less serious than spinal affections. In the spinal cord the centres and conducting paths occupy so small a space transversely that a comparatively slight lesion may injure it irretrievably; while the brain may

be the seat of relatively large lesions, without any definite symptoms being produced.

The prognosis in a particular case often depends to a large extent upon the cause of the affection. In the rheumatic, toxic, syphilitic, and hysterical affections, and in those which occur after diphtheria and other acute diseases, the prognosis is on the whole favourable. The prognosis in hæmorrhage depends upon its extent and the locality of the lesion. Hæmorrhage into the substance of the spinal cord, unless it be very limited, is most unfavourable; while cerebral hæmorrhage, unless of large extent, is favourable so far as partial recovery is concerned. Almost complete restitution of the functions may take place provided the fibres of the motor tract are uninjured; but rupture of these fibres is followed by descending changes in the pyramidal tract and permanent paralysis with contracture of the affected extremities. The prognosis in cases of embolism and thrombosis depends upon the size of the vessel obstructed, upon whether collateral circulation can be readily established or not, and upon the functional importance of the part to which the vessel is distributed, obstruction of the vessels supplying the motor mechanism giving rise to more definite and permanent symptoms than obstruction of the vessels of other parts.

Very little can be said with regard to prognosis in inflammatory affections of the nervous system. Acute and severe inflammatory attacks are often either fatal or give rise to incurable symptoms. The prognosis in subacute inflammations is often favourable; while that of the chronic inflammations and degenerations is always unfavourable so far as complete restitution is concerned; although they may extend over a long lifetime without producing symptoms which endanger life. Inflammation of grey matter is probably more immediately dangerous to life than that of the white substance; but if the danger of the primary symptoms is overcome the probability of ultimate recovery is much greater in the former than in the latter. Acute inflammations of the central grey substance of the spinal cord are almost uniformly fatal; but the prognosis is by no means unfavourable in subacute inflammations. And although inflammation of the white substance of the cord is not so immediately



dangerous to life, the probability of complete recovery is less than with inflammation of the grey substance. When the pyramidal tract is injured either in the cord or in its course through the brain the probability of complete recovery is small. A certain amount of degeneration of the tract, or loss of conduction through it from compression, may be recovered from; but when some of its fibres are completely ruptured by a neighbouring hæmorrhage or other cause, or when degeneration of the fibres has lasted a considerable time, complete recovery from the paralytic symptoms must not be expected. When, therefore, the paralysed muscles are for some little time the subjects of muscular tension, contractures, and exaggeration of the deep reflexes, complete restitution is impossible.

In the atrophic forms of paralysis, whether of spinal or peripheral origin, much valuable aid in forming a prognosis is obtained from electrical examination of the affected muscles. If the faradic and galvanic contractility of the muscles is very slightly diminished without any qualitative changes, recovery will be rapid. Even when the "reaction of degeneration" is present, and the faradic contractility of the affected muscles is abolished for a time, if slight voluntary movement and faradic contractility reappear at the end of six weeks from the onset of the affection, recovery will be comparatively rapid. If, however, slight motor power and the faradic contractility do not reappear until the thirtieth week from the onset of the disease, recovery will be very slow (extending over a period of twelve months), and often imperfect. When every trace of both faradic and galvanic irritability has disappeared from the muscle, recovery is no longer possible.

The prognosis in paralysis of whatever kind becomes more grave when it is accompanied by well-marked anæsthesia and by decided vaso-motor and trophic disturbances, especially by extensive and sloughing bed-sores. In spinal paralysis the prognosis becomes very grave when the sphincter of the bladder and rectum become paralysed, and when the urine is alkaline and fœtid and contains pus; while commencing paralysis of the muscles of respiration and of deglutition indicates impending dissolution.

## CHAPTER IX.

## GENERAL TREATMENT.

IN the management of nervous diseases the organism, as a whole, must be subjected to treatment. Consequently the rules to be observed in the treatment of the nervous system are essentially the same as those which experience has proved to be useful in the treatment of diseases of other tissues and organs of the body. It is therefore unnecessary to describe, in detail, the principles which ought to guide us in the treatment of diseases of the nervous system.

The treatment of the nervous system may be divided into that which is directed : (1) to prevent disease (prophylactic) ; (2) to remove the exciting cause of the disease ; (3) to remove the anatomical cause ; and (4) to allay or remove serious symptoms.

§ 132. (1) *Prophylaxis*.—The prophylactic treatment of disease may, to some extent, be regarded as part of general hygiene. It consists, indeed, of a special application of hygienic rules to the cases of those who manifest inherited or acquired proclivities to certain diseases of the nervous system. A young lady, for instance, whose mother has suffered from severe trigeminal or other form of neuralgia, should be specially cared for, and protected from mental excitement and unnecessary exposure to cold, at the period of sexual development, when neuralgia is so apt to become established for the first time. The children of parents who manifest a predisposition to severe nervous disease, as hysteria and epilepsy, are frequently not merely quick in their perceptive faculties, but are also often possessed of great intellectual powers, and much of their future



happiness depends upon judicious mental training in youth. Even the children of families, some members of which have suffered from the graver psychoses, are no exceptions to this rule, as it is notorious how often "genius to madness is allied." The children of such families ought not to be subjected to any severe mental strain during the period of bodily development, or allowed to enter into competition with other children in the mental gymnastics which are so fashionable at our public schools. On the other hand, regular, graduated, and systematic exercise, in the form of walking, riding, gymnastics, and calisthenics, does a great deal of good by strengthening both the muscular and nervous systems. Everything which tends to develop the muscles of the lower extremities and trunk, and, indeed, all muscles engaged in executing the movements common to both man and the lower animals, tends also to develop the fundamental part of the nervous system; and a good sound development of the fundamental is the first pre-requisite to a well balanced development of the accessory portion.

The order of the development of the nervous system in the race has been from the fundamental to the accessory portions, and no one can reverse this process with impunity in that further development of the individual which constitutes education in its widest sense. Yet until a few years ago the natural order of development was reversed in the education of youth, and especially in female education, so far as this could be accomplished by human contrivance and ingenuity. The natural order of development was indeed observed so far as to allow the child to acquire the power of walking prior to that of other accomplishments, but the care of the infant had not yet been transferred to the professional trainer. No sooner, however, had what is technically called education begun than the professional trainer began to exercise the small muscles of vocalisation and articulation so as to acquire the art of reading, the small muscles of the hand so as to acquire the art of writing, and in the case of young ladies the still more complicated movements necessary in running over the key-board of a piano; while little attention was paid to the development of the larger muscles of the trunk and lower extremities upon the full development of which the future health and comfort of the individual depends.

In the education of youth, in the present day, the laws of development and physiology are not so openly violated and defied as they were a few years ago; but much yet remains to be done in this respect, and especially in the education of the children of families who manifest a neuropathic tendency. In the children of such families the greatest possible care should be taken to develop carefully the fundamental actions, inasmuch as a sound development of these involves a stable construction of the fundamental part of the nervous system, a process which enables the latter to offer greater specific resistance to the paroxysmal discharges from the later evolved centres of the accessory portion which underlie hysteria, epilepsy, and even many of the psychoses. The process of educating the accessory system, and especially the higher centres of that system in young people with a neuropathic predisposition, should be regular and systematic; habits of mental scrutiny and self-examination, which, unfortunately, too many religious teachers deem necessary for the welfare of the soul, ought to be discouraged. In one word, education should be made as concrete and objective as possible. By such training it is not only possible to gain access to the highest treasures of the intellect, to poetry, music, and art, but such training can also be made subservient to the due development of the religious sentiments. Sentimental novels, with harrowing descriptions of lacerated feeling and wounded vanity, are to be utterly proscribed; but novels like those of Scott, which lead the mind outwards to contemplate natural scenery, historical incidents, and the manners and mode of living of a past age, are really useful educational adjuncts. Fatigue, late hours, undue excitement from theatres and balls, or from the use of alcoholic stimulants, and every other means by which the nervous energy is exhausted, ought to be carefully avoided. It ought to be mentioned that young people with a neuropathic predisposition are peculiarly liable to indulge in sexual excess; and their guardians should, therefore, be specially on the alert lest they indulge in the practice of onanism, which exercises a most deleterious influence on the nervous system, both strongly predisposing to various diseases, and acting as a powerful exciting cause of grave nervous disease.



It is scarcely necessary to add that those who inherit a predisposition to nervous diseases require, in addition to exercise, careful regulation of diet and a due exposure to sunlight and fresh air. In many cases the administration of iron and cod liver oil is attended with the most beneficial results; in one word, the standard of the health must be maintained at its highest point of efficiency, and this can be done, not by treating the nervous system, but by treating the organism as a whole. The beneficial effects of rest, both in the prevention and treatment of disease, are now so well recognised, that it is unnecessary to dilate upon the subject in this place.

§ 133. (2) *Removal of the Exciting Cause.*—The next indication of treatment is to remove the exciting cause of the disease, and it is quite unnecessary to say much at present on this subject. If the disease has been induced by unfavourable conditions of climate, exposure to variations of temperature, excessive fatigue, these conditions must be obviated. When the affection has been caused by wounds and contusions of the nervous tissues, or by compression, as by the gradual growth of tumours, the exciting causes must be removed by surgical interference, and the damage done to the tissues treated according to general surgical principles.

If the disease is caused by a morbid poison, as syphilis malaria, gout, rheumatism, and the metallic poisons, the treatment must be first directed to remove these poisons from the system or to neutralise their action. Syphilitic disease of the nervous system occurs in so many forms that it may be said to simulate almost every form of grave nervous disease. But whatever may be the form in which it appears, the same treatment must be adopted as for syphilis of other tissues and organs. It must be remembered, however, that syphilitic disease of the nervous system usually belongs to the late or tertiary symptoms of the affection, and consequently the treatment must be modified accordingly. Syphilis of the nervous system as a rule demands large doses of the iodide of potassium, not less than from a scruple to half a drachm three times a day; and if this fail to give relief, from one-twelfth to one-eighth of a grain of the bichloride of mercury may be given three times a day.

In neuralgia and other diseases of malarial origin quinine and arsenic are the best known and most reliable remedies. In neuralgia and other diseases of gouty origin a moderate dose of colchicum along with a saline aperient is useful; while rheumatic neuroses are benefited by salicylic acid, local fomentations, vapour bath, and in chronic affections, by the use of iodide of potassium and guaiacum.

In acute poisoning a drug may at times be administered whose action counteracts that of the first agent. Atropine, for example, appears to arrest to some extent the poisonous action of opium, chloral that of strychnine, and this list might be indefinitely extended; but the practical results which have been obtained from the administration of the so-called antidotes have never realised the high expectations of their utility which was at one time formed.

§ 134. (3) *Removal of the Anatomical Cause.*—The anatomical cause of diseases of the nervous system must be removed by favourably influencing the nutrition of the diseased part, no matter whether the lesion be molar or molecular. The nutrition of the nervous system as a whole and of its various parts may be influenced in various ways. Nutritive changes may be produced by agents which act directly on the nervous tissues themselves, the connective tissues surrounding the nervous elements including the lymph spaces, the blood-vessels including their vaso-motor nerves and centres, or the blood. The agents which act on each of these tissues may in practice be variously combined; so that the methods by which the nutrition of any part of the nervous system may be influenced are almost innumerable. We shall not attempt any elaborate classification of the remedial agents comprised in this division further than is implied in an orderly enumeration of some of the more important of them, but shall found our confidence in them almost entirely on empirical observation.

The agents comprised in this subdivision may be subdivided into: (A) Internal Remedies, (B) External Remedies:—

#### (A) INTERNAL REMEDIES.

Internal remedies are those which act after being absorbed



into the blood, no matter whether they are absorbed through the stomach, the skin, after subcutaneous injection, or are directly injected into a vein. The following are some of the remedies of this class which are most employed in the treatment of the diseases of the nervous system.

§ 135. *Strychnine*, and the preparations of *nux vomica*, increase the irritability of the grey substance of the spinal cord and consequently diminish its resistance, and they are usefully administered when the spinal irritability is depressed. These agents produce the most favourable effect in cases of muscular weakness unattended by organic lesion. Their administration is attended probably with more benefit in the visceral than in the external akineses. These agents are powerful remedies in atonic dyspepsia, constipation with flatulence, paralysis of the sphincters, nocturnal incontinence of urine, sexual debility; and Dr. Milner Fothergill has strongly advocated the administration of strychnine as a stimulant of the respiratory centres in cases of emphysema and bronchitis. Strychnia is not, however, an agent of much value in the treatment of nervous diseases depending upon structural change. In all acute affections of the nervous system it is positively injurious; while in the chronic organic diseases it is almost valueless.

*Conium* acts in some respects in an opposite way to strychnine by depressing the reflex irritability of the cord. The investigations of Drs. Crum Brown and Fraser prove that the alkaloid *conia* produces paralysis solely by influencing the motor nerves, and is in this respect analagous in its action to curara. They have, however, shown that some of the methyl compounds of conia depress the irritability of both the motor nerves and of the spinal cord. These authors have likewise found that commercial specimens of conia are a mixture in variable proportions of conia and methyl-conia, and that the succus conii of the Pharmacopœia also contains methyl compounds of conia; so that conia as usually administered depresses the irritability of the spinal cord, although the pure alkaloid only acts on the motor nerves. Conia has been administered successfully in cases of tetanus, but it is probable that the beneficial results which followed its administration were due to

the impurity of the drug, and that more reliable results would be obtained by the use of hydrochlorate of methyl-conia. Conia does not appear to exercise any influence on the action of the heart. Dr. Crichton Browne strongly recommends conium in acute mania, and, whatever may be its mode of operation, experience shows that it is a powerful agent in allaying excitement under such circumstances.

§ 136. *Calabar Bean* gradually lessens and ultimately destroys the irritability of the grey substance of the cord, causing anaesthesia, loss of reflex excitability, and paralysis. Its action is therefore more directly opposed to that of strychnine than that of methyl-conia. Calabar bean, or its alkaloid physostigma, may therefore be administered in cases of increased irritability of the grey substance of the cord; such as that which occurs during strychnine poisoning, and this agent has been successfully employed in the treatment of tetanus (Watson, Ringer). Physostigma diminishes the number of the heart's contractions, but lessens the duration of each systole, and at last the heart ceases to beat in diastole. During this action a weaker electric current through the vagus is required to arrest the action of the heart than in health, and if the ends of the pneumogastric nerves are poisoned by atropia, physostigma will restore the action of the paralysed nerve; hence it has been concluded that this drug increases the irritability of the terminal fibres of the pneumogastric nerve. This agent is also a respiratory poison, and generally kills by paralysing the respiration.

§ 137. *Atropine, or Belladonna*, is a powerful agent in the treatment of many diseases of the nervous system. The following is a brief summary of the actions of atropia. It increases the irritability of the grey substance of the spinal cord, acting specially on the respiratory and vaso-motor centres, and also stimulates the cardiac acceleratory nerve or its centre. It paralyses the motor nerves, first affecting those of the trunk, the terminations of the vagi both in the heart and lungs, the terminations of the secretory nerves of the salivary glands and of the sweat glands, the terminations of the inhibitory fibres of the splanchnics, and the terminations of the nerves



supplying the iris. It appears also to stimulate the pupillary fibres of the sympathetic nerves to the eyes. In large doses it depresses the functions of the afferent nerves. The therapeutic uses of belladonna are so manifold that we can only mention a few of them at present. This drug has been found useful in checking profuse sweating, especially the night sweats of phthisis; and the secretion of milk. It is also very useful in habitual constipation, and has been recommended by Harley as a cardiac tonic. The action of atropia is antagonistic to opium, calabar bean, muscarin, jaborandi, bromal, aconite, and prussic acid (Ringer). Belladonna is exceedingly useful in allaying the paroxysmal cough of whooping cough, and probably also diminishes secretion in cases of chronic bronchial catarrh. It is one of the best remedies in cases of incontinence of urine, and, combined with zinc, it is often a successful remedy in cases of nocturnal emissions (Ringer). Its use was recommended by Dr. Brown-Séquard in the treatment of paralysis depending upon chronic inflammation of the spinal cord, on the supposition that it produces contraction of the arterioles of the cord, and thus diminishes the supply of blood to it. Belladonna is, no doubt, useful at times in the treatment of chronic spinal affections, but it is doubtful how far the theory upon which this treatment was originally founded is correct. Belladonna has also been found useful in the treatment of epilepsy. In addition to its action on the spinal cord and peripheral nerves, belladonna also acts on the brain. The first effect is stimulant, giving rise to a rapid but connected succession of ideas; but the ideas soon become extravagant and incoherent, and a busy delirium sets in accompanied by pleasing illusions. The delirium may at times be furious and dangerous, so that the patient requires to be placed under restraint. Its action on the cerebrum renders belladonna a valuable agent in allaying pain.

§ 138. *Opium* lessens the irritability of the sensory conducting paths and of the perceptive centres. Small doses first increase the irritability, but the primary increase is soon followed by a secondary stage of depression, and if a large dose be administered the first stage of increased irritability is so transitory that it may be entirely overlooked. Bernard concluded that

opium also depresses the irritability of the sympathetic system, and especially the part supplying the submaxillary gland. Opium may be administered, like all agents of this class, in small doses, with the view of obtaining its primary or stimulant action, and in large doses so as to obtain its secondary or sedative action. In small doses it is found useful whenever the irritability of the nervous system is depressed, especially during times of nervous exhaustion produced by anxiety and overwork. In larger doses this drug is found useful whenever the irritability of the sensory mechanism is increased. Opium and its alkaloids are indeed the most powerful agents we possess for allaying pain and procuring sleep, and are therefore our most generally useful aids in removing the most distressing symptoms of nervous diseases.

§ 139. *Hydrate of Chloral* in moderate doses induces sleep, and in large doses profound coma. Both sensation and reflex action is diminished under its action, and death is caused by arrest of respiration or paralysis of the heart. Dr. Hammond suggested that the action of chloral is due to anæmia of the cerebral substance, caused by vaso-motor contraction of the vessels of the brain; but it is much more probable that chloral, as well as chloroform, ether, and the other agents which are called anæsthetics, act directly on the nervous tissues themselves. All these agents have, like opium, a primary stimulant, followed by a secondary sedative action. They appear, indeed, to act as poisons to any living protoplasm, although the phenomena produced by arrest of the special functions of the nervous tissues becomes so marked and predominant as to obscure the effect on the protoplasm of the other tissues.

When the agents which are classed as anæsthetics are introduced into the circulation, the rapidity with which each will reach the protoplasm of the tissues will depend on the degree of diffusibility of the drug, the activity of the circulation in a part, and the density of the cell walls and intercellular substance of the tissues. The more diffusible the agent the sooner it gains access to the tissues, and the sooner it is eliminated; hence the greater the diffusibility of the agent the sooner is the action produced, but the more transitory will the action be.



Those tissues which are supplied with a large quantity of blood will also have relatively a large quantity of the drug supplied to them; hence the active tissues will be affected before the passive tissues, and the grey substance of the brain will be affected in preference to the white substance. The protoplasm of the tissues which are composed of small cells with thin cell-membranes will be reached by the agent in a shorter time and in a larger quantity than that of tissues consisting of large cells with denser cell-membranes; hence the small cells of the sensory mechanism will be affected in preference to the large cells of the motor mechanism.

Chloral and allied agents are usually administered in relatively large doses with the view of depressing the irritability of the sensory mechanism and procuring sleep. Chloral has been found useful in the treatment of tetanus and of strychnine poisoning, and as a sedative and hypnotic in cases where the irritability of the sensory mechanism is increased.

§ 140. *Bromide of Potassium* and its allies depress the irritability of the brain and spinal cord, inducing in large doses a diminution of sensibility and of reflex excitability. Its action is, indeed, like that of chloral; although the action of the latter is more prompt yet more evanescent. It is very probable that the greater atomic weight of the constituents of bromide of potassium, as compared with those of chloral, renders the elimination of the former from the body more slow and difficult than that of the latter. The bromide of potassium also appears to lower the irritability of the motor mechanism, while chloral acts more exclusively on the sensory mechanism; but both drugs seem to act on the grey centres of the brain and spinal cord in preference to the conducting paths. Bromide of potassium is a most valuable remedy when it is desired to depress more or less permanently the irritability of these grey centres; while chloral is the better remedy when a prompt but temporary action is required. Bromide of potassium is therefore a more valuable remedy in hysteria and epilepsy, and in various other spasmodic affections.

§ 141. *Iodide of Potassium* has probably no special action

on the nervous tissues themselves, but its well-known action in quickening the absorption of inflammatory effusions renders it an invaluable agent in the treatment of many nervous diseases. Its use in syphilitic nervous affections has already been mentioned.

§ 142. *Mercurial Preparations* are also occasionally useful in the treatment of inflammatory diseases of the nervous system, more especially those affecting the membranes of the brain and spinal cord and the sheaths of nerves; but their use in the present day is in a great measure restricted to the treatment of syphilitic nervous diseases.

§ 143. *Zinc* and its preparations belong to a class of agents which have been termed "nervine tonics," on the supposition that they promote the nutrition of the nervous system. It is probable that these agents enable weakened nervous tissues to absorb a larger stock of nutriment from the blood, while at the same time the molecules of these materials are so arranged that greater resistance is offered to change, hence external stimulation does not give rise to such wide-spread nervous discharge, while the resulting local discharges are more powerful and better co-ordinated. But it will suffice for us to know at present that these agents are proved by experience to exercise a favourable influence on the nutrition of the nervous system. Preparations of zinc have been successfully used in the treatment of chorea, epilepsy, whooping-cough, and other spasmodic affections.

§ 144. *Arsenic* is, according to Dr. Ringer, a protoplasmic poison, destroying first the functional activity of the central nervous system, next of the nerves, and last of the muscles. But like all other poisons of this class, it is probable that the first effect of small doses is to stimulate the functional activity of protoplasm, and that its administration in regulated doses will be beneficial in many cases of nervous debility. It has been found useful in some chronic diseases of the spinal cord, and is certainly a powerful remedy in many cases of neuralgia.

§ 145. *Phosphorus* is another agent which has, of recent years, been strongly recommended in the treatment of various



nervous affections. It probably acts as a stimulant to the nervous centres, and increases the nutritive activity of nervous tissues generally. It has been found useful in functional paralysis, chronic alcoholism, migraine, and in many forms of neuralgia.

§ 146. *Silver* salts have been employed in the treatment of nervous diseases. Nitrate of silver in poisonous doses excites convulsions in animals, and these are followed by paralysis. The convulsions are similar to those produced by strychnia, and are excited by the least peripheral irritation (Ringer). Both the oxide and nitrate have been employed in the treatment of chorea and epilepsy, and they have been found specially useful in the treatment of locomotor ataxy.

§ 147. *Quinia* is one of the most generally useful nerve tonics. Administered in large doses it produces noises in the ears, and occasionally causes deafness, and even temporary blindness may be produced. It also causes severe dull frontal headache, and in poisonous doses abolishes reflex action before voluntary movement, by stimulation of Setchenow's reflex inhibitory centre (Ringer). The best known action of quinia is that by which it arrests attacks of ague; but it is equally useful in the treatment of other affections of malarial origin. Neuralgia is especially prone to arise from malaria, and against this form of the disease quinia is a specific. But quinia is also useful in other forms of neuralgia, especially when they assume a periodic type, as frequently happens in neuralgia of the supra-orbital branch of the fifth nerve.

§ 148. *Milk, whey, and grape "cures,"* as well as courses of *mineral waters*, produce a beneficial effect on diseases of the nervous system, in so far as they exercise a favourable influence on the nutrition of the body generally.

#### (B) EXTERNAL REMEDIES.

§ 149. *Cold*, steadily applied, lowers the irritability and retards the conductivity of the nervous tissues. Riegel and F. Schultze have shown that the spinal cord can be directly

reached by the application of cold; but the experiments of Dr. Benham appear to indicate that the local influence on the brain, of cold steadily applied to the head, is not great. Chapman's vaso-motor therapeutics is founded on the assumption that cold applied to the spine lowers the irritability of the vaso-motor, along with that of the other centres, of the spinal cord, and thus causes dilatation of the blood-vessels, whose nervous supply issue from the portion of the cord over which the cold is applied. Chapman's ice bag is the best method of applying cold to the spine; and very convenient bags are also constructed for applying ice to the head, or, indeed, to any part of the body.

§ 150. *Warmth* increases the irritability, and accelerates the conductivity of the nervous tissues. It also relaxes the tissues, especially when moisture is combined with warmth, and it is probably by this means that it exercises such a soothing influence in painful and spasmodic affections. It is doubtful whether warmth directly applied over the head and spine penetrates to the brain and spinal cord; but warm applications are often very soothing in affections of these organs, the action being probably produced in a reflex manner through the cutaneous nerves. Warmth may be applied by means of hot water fomentations, poultices, Priessnitz's compresses, hot sand bags, or caoutchouc bags filled with hot water.

§ 151. *Baths*.—Cold baths tend directly to lower the irritability of the nervous tissues, and to depress the nervous functions; and when their use is attended with benefit it is due to their indirect action. The first apparent effect produced by a cold bath is to contract the cutaneous blood-vessels, but whether this effect is produced by a reflex stimulation or by a direct action on the muscular fibres and peripheral terminations of the nerves is not known. Contraction of all the superficial blood-vessels is followed by a sudden rise in the arterial tension, the beat of the heart becomes more powerful, and the internal organs, including the great nervous centres, are more freely supplied with blood. The increase in the supply of blood to the nerve centres raises their irritability, so that more powerful nervous discharges are



sent out to the various organs, including the heart, rendering its action more effective, and thus increasing still further the activity of the circulation.

However efficient the cold bath may be as a means of treatment in many cases of nervous debility and temporary exhaustion, it is manifest that a pre-requisite to the success and safety of the treatment is that the patient be practically free from any organic disease. If, for instance, the central organ of the circulation be enfeebled from any cause, the sudden elevation of the arterial tension may arrest its action; if the walls of some or all of the blood-vessels have lost their elasticity and are become brittle from degenerative processes, rupture may ensue; or if the walls of the vessels of any internal organ, and especially if one or more of the nervous centres, are enfeebled by local inflammation or other disease, a local congestion may be caused which will greatly aggravate the affection. The cold bath is, however, a powerful therapeutic agent in the numerous functional diseases to which the nervous system is liable; and, notwithstanding the drawbacks and limitations which have just been described, it is also, with suitable precautions, useful in the treatment of various chronic organic affections.

§ 152. *The Sea Bath* is only a modification of the cold bath, but several factors combine to make the action of the former somewhat different from that of the latter. The salts in solution in sea-water act as powerful stimulants to the skin; hence sea-water of itself is much less depressing than ordinary water, so that feeble persons who cannot bathe in the latter without suffering great depression can bathe in the former with benefit. The motion of the waves, however, has a powerfully depressing effect; but the reaction following from the dashing of the waves against the body adds greatly to the invigorating and exhilarating effects in strong and healthy persons.

If the nervous system of the patient is so weak that the necessary reaction does not take place, even although there may be no organic disease, outdoor sea-bathing must be prohibited, but an indoor bath of sea-water, either cold or with the chill taken off, may be substituted. A considerable part of the benefit received by patients from residence at the sea-side must be ascribed to the breathing of sea-air, along with the complete change of scene and occupation.

§ 153. *The Cold Water Pack* is a powerful therapeutic agent, but it is seldom employed outside hydropathic establishments, and there it is associated with a carefully regulated diet, change

of air and scene, regular hours, and various other circumstances which assist reparative processes ; so that it is difficult to determine how much of the beneficial effect is due to the cold water treatment. There can be no doubt, however, that the cold water pack, along with the subsequent sponging and rubbing, is exceedingly refreshing, and under its use tissue metamorphosis is accelerated, the nutrition of the body is improved, and the absorption of effused products is promoted ; hence it becomes a valuable remedy in many organic nervous diseases.

§ 154. *Shower, Douche, and Sponge Baths* produce, in addition to the effects of the cold immersion, powerful nervous stimulation from the impact of the water on the body.

Shower and sponge baths are useful in cases of hysteria and other functional nervous diseases, while the douche and cold affusion are generally employed for their local effects, as, for example, to rouse a patient from drunkenness or opium poisoning. The local douche has also been found useful in the treatment of paralysed parts, and in several forms of sensory disturbance. A douche, consisting of alternations of hot and cold water, has a powerfully stimulating effect, and may be employed in many cases of paralysis.

§ 155. *Warm Baths*, by sheltering the body from the constant changes of the external air, and by diminishing oxidation, act as a sedative to the cutaneous nerves, and through them to the nervous system generally. By procuring dilatation of the cutaneous blood-vessels, arterial tension is lowered, and relief is thus afforded to congestion of internal organs. The effects vary considerably, according to the temperature of the bath. If the temperature be indifferent ( $90^{\circ}$  to  $97^{\circ}$  F.) the bath acts mainly as a nervous sedative, while warm and hot baths ( $97^{\circ}$  to  $108^{\circ}$  F.) produce great vascular excitement, and act as powerful nervous stimulants.

The geographical position of the bath must be taken into account, especially as experience teaches that the higher the elevation, the higher is the temperature that can be borne ; and that the more irritable the patient is, the more elevated may be the spot to which he is sent for cure (Erb). When the symptoms of irritation preponderate we choose the soothing bath ; and if the symptoms of depression are prominent, the



exciting bath at a higher temperature may be employed. A complete list of hot springs, and much useful information with respect to spas and mineral waters generally, are given in Squire's Companion to the British Pharmacopoeia; and the information given in a work which is in the hands of almost every practitioner need not be repeated here.

§ 156. *Weak Brine Baths*, containing not more than one per cent of chlorides, most sulphur baths, and the weak alkaline springs act like indifferent springs, and may be employed in the treatment of the same class of diseases. These baths are useful in spinal irritation and exhaustion from excess of any kind, and in spinal meningitis; but the chronic scleroses of the spinal cord require great care in the use of the warm bath, inasmuch as cases of locomotor ataxy and chronic myelitis are often made worse by too warm baths.

§ 157. *Vapour Baths, Hot Sand Baths, Hot Air Baths* produce diaphoresis, and are powerful nervous stimulants, and may be used in the treatment of the same class of cases as the hot spring baths.

§ 158. *Brine Baths* differ only from warm baths from the fact that the contained salt acts as a powerful stimulus of the nutrition and circulation of the skin; and on account of the exciting effect of the salt the temperature may be somewhat lower than that of warm baths. The most suitable proportion of salt is from two to four per cent (Erb).

§ 159. *Warm Brine and Gaseous Baths* are more stimulating than the simple brine baths, inasmuch as the carbonic acid contained in these baths powerfully excites the skin and nervous system. These baths should not exceed 90° F., and they are usually taken without moving the water, but when a strong stimulant is desirable the water may be agitated. These baths are indicated in the chronic stage of atrophic paralysis, in locomotor ataxy, and other chronic affections of the spinal cord, as well as in cases of functional debility of the nervous centres.

§ 160. *Chalybeate and Gaseous Baths* appear to act precisely in the same manner as the alkaline and gaseous baths. The iron does not appear to be of any value except when used internally.

§ 161. *Mud Baths* act partly as warm water baths, but they are much less exciting than warm water spring baths, and are consequently adapted for the treatment of spinal irritation and paralysis accompanied by excess of muscular tension, as occurs in lateral sclerosis, compression of the cord, and hemiplegia with descending sclerosis. Good mud baths are found in Franzensbad, Marienbad, Teplitz, Driburg, Eilsen, Meinberg, Nenndorf, Pyrmont, and other places.

§ 162. *The Turkish Bath* combines many of the properties of the hot and cold bath. It produces profuse diaphoresis, and thus cleanses the system by carrying off effete and noxious substances, so that its use is indicated in many nervous affections which occur in rheumatic and gouty constitutions. The great heat to which the body is subjected tends no doubt to induce debility; but the subsequent free application of cold water excites the cutaneous nerves and braces the system, so that the tonic effects of the cold bath are procured. The systematic kneading to which all the muscles of the body are subjected promotes their own nutrition and the nutrition of the nerves which supply them, so that the effects of the bath are combined with those which result from carefully-regulated exercise and systematic gymnastics. The Turkish bath is, indeed, a powerful therapeutic agent, and has a wide range of usefulness in the treatment of various nervous affections.

§ 163. *The Needle Bath* consists of an apparatus so constructed that numerous jets of water forcibly impinge on the body of the patient. Hot water is first used, and after a few minutes it is gradually or somewhat suddenly changed for cold water. The alternation of hot and cold water, as well as the impact of the jets against the body, renders this bath a powerful stimulant, and it is exceedingly useful in the treatment of chronic nervous diseases. It is a very valuable means of treatment in loco-



motor ataxy, for instance, when the lightning pains are not excessive, and when the tendinous reflexes are abolished. In lateral sclerosis, when the reflexes are increased, this bath, like all other stimulating baths, does much harm.

§ 164. *Climate "Cures."*—It has been found by experience that certain climates and regions exercise a favourable influence on various nervous diseases, especially those of functional origin. Sea air has a very invigorating effect on the system, and it is well adapted for the cure of states of nervous exhaustion from overwork in those who are otherwise strong and healthy. Feeble and irritable people, however, obtain greater benefit from residence in a mountainous district. Mountain exercise has a very enlivening effect upon the nervous system, and the higher and drier the district the more marked are the tonic effects. The Engadine and Davös Platz are very favourite mountain residences; and the Scottish mountains, the English lake district, and Ilkley in Yorkshire, are also beneficial, although they have neither the elevation nor the dry atmosphere of the Swiss mountains.

§ 165. *Blood-Letting and Counter-Irritation* are as useful in the treatment of inflammatory affections of the nervous system as in those of other organs, but as they present nothing special in their action or mode of application when employed in the treatment of nervous diseases, they do not require further consideration at present. The counter-irritants usually employed are cutaneous faradisation, sinapisms, vesicants, issues, moxæ, and the actual cautery.

§ 166. *External Frictions* are usefully employed in various nervous affections. Frictions with soothing liniments, warm oil, opium, and belladonna ointment, and various other agents, are used to allay pain in neuralgic and other painful affections. Frictions with spirituous liniments, either alone or combined with other stimulants, such as ammonia and camphor, are used in the treatment of various forms of paralysis.

§ 167. *Massage* is a general term meant to include methodical rubbing, stroking, kneading, and clapping the surface of feeble

and paralysed parts; a method of treatment which is often successful in the treatment of both functional and organic paralysis.

§ 168. *Swedish Gymnastics* are a mere modification of ordinary gymnastics, with the view of aiding the recovery of paralysed or rather paretic parts, by bringing the muscles into methodical exercise. The patient is instructed to make systematic voluntary efforts to move the paralysed muscles, and when a certain degree of voluntary movement is attained the effect is increased by the opposition of a practised assistant. If, for example, the flexors of the forearm are to be exercised the forearm is first extended, and the patient endeavours to flex it whilst the assistant opposes flexion with more or less force. In the case of associated movements it is often necessary to aid the contraction of one group of muscles so as to overcome the contraction of their antagonists, which are also excited to action during the voluntary effort to contract the paralysed group. The assistant must then aid the paralysed muscles by passively extending the healthy antagonist muscles. The action of the healthy antagonist muscles can be weakened, not only by the hands of an assistant, but also by fixing elastic bands or spiral wires to the limb so as to aid the action of the paralysed groups. Ordinary gymnastic exercises are also useful in the treatment of various nervous affections. By their use the nutrition of the muscular system is improved, the heart propels the blood more efficiently through the organism, the blood itself becomes of better quality, and the nervous system participates in the general improvement.

§ 169. *Electricity* in its various forms is not only one of the most useful remedies we possess for the treatment of disease of the nervous system, but it is also a remedy which is almost exclusively adapted for the treatment of nervous disease. The therapeutical applications of this agent, therefore, demand from us a somewhat lengthened examination. The varieties of electricity are—(I.) *Franklinism*; (II.) *Faradism*; and (III.) *Galvanism*. The act of applying electricity in the treatment of disease is called *Electrisation*, while the acts of applying its varieties are respectively called *Franklinisation*, *Faradisation*, and *Galvanisation*. For a description of the various instru-



ments used in applying electricity we must refer the reader to systematic works on electro-therapeutics, and to all respectable surgical instrument makers, who now keep in stock reliable medical electric machines with their accessories.

(I.) *Franklinism, Friction, or Static Electricity*, is generated in the usual way by a plate machine.

### § 170. *Methods of Application.*

(1) *Electro-Positive Bath*.—The patient must be insulated by being placed on a glass-legged stool or couch, and then connected by a brass chain, held in his hand, with the prime conductor of the machine. When the plate of the machine is rotated both the conductor and the patient become charged with positive electricity, hence the patient is said to be taking an *electro-positive bath*.

In order to prevent the moisture of the atmosphere from carrying off the charge, the plate, the insulating supports, the legs of the stool, and all the glass part of the apparatus should be well rubbed with a warm and dry piece of flannel.

(2) *Electro-Negative Bath*.—If the chain held in the hand of the patient be connected with the cushions of the machine, instead of with the conductor, he becomes charged with negative electricity, and is said to be taking an *electro-negative bath*.

(3) *Franklinisation by Sparks*.—If, when the patient is in connection with the prime conductor, any object be brought near to him, he is *discharged* with a spark; the discharge is accompanied by a slight shock, and is called *Franklinisation by sparks*. The electricity may be localised to some extent without producing a shock by passing a metallic brush slowly along the course of a nerve trunk or a muscle, almost but not quite in contact with the skin.

(4) *Franklinisation by the Leyden Jar*.—A Leyden jar is charged in the usual way, and is discharged by applying the extremities of the excitors to two points of the body through which it is desired the electricity should pass. Franklinisation is not a method of treatment of which I have personally much experience; but Dr. Tibbits speaks favourably of it as a remedy in neuralgia, sparks being drawn along the track of the affected nerves; also in facial spasm, emotional aphonia, hysterical hyperæsthesia, and various forms of tremor.

(II.) *Faradism*.—The *induced or interrupted current*, as it is variously called, was discovered by Faraday, and is generated or induced in a coil of copper wire by the action of a permanent magnet, or of a voltaic current. The instruments constructed to generate the faradic current possess two coils, called the *primary*

and *secondary* coil, from each of which a current is obtained; these are respectively called the *primary induced* and the *secondary induced current*. The current induced in the secondary possesses greater tension than that of the primary coil; hence the former penetrates more readily through the tissues than the latter, and consequently acts as a more efficient stimulus to the nerves and muscles.

### § 171. *Methods of Application.*

(1) *General Faradisation*.—Drs. Beard and Rockwell, who introduced this method, recommend that the patient be placed with his naked feet upon a sheet of copper, which is connected with one pole of the battery. The operator holds the other pole, containing a well moistened sponge, in his left hand, and passes the right hand over the muscles or whole body of the patient. The authors think that general faradisation stimulates both local and general nutrition, and thus acts as a powerful constitutional tonic.

(2) *Localised Faradisation* is, as its name implies, the application of the faradic current to special organs or tissues. This mode of application admits of the following subdivision:—(a) Cutaneous faradisation; (b) Neuro-muscular faradisation; and (c) Faradisation of internal organs and of the special senses.

(a) *Cutaneous Faradisation*.—When it is desired to limit the current to the cutaneous surface the rheophores must be used dry, and the skin should also be dusted with some absorbent powder, so as to diminish its conduction. Besides the usual rheophores other methods are adapted for applying cutaneous faradisation.

(i.) *The Electric Hand*.—A moist rheophore is applied to some slightly sensitive part of the patient's body, as over the sternum; the other rheophore is held in the hand of the operator, who passes the back of the disengaged hand over the surface which it is desired to excite.

(ii.) *Solid Metallic Rheophores* are applied by thin metallic surfaces to the dry skin, and are either kept stationary or stroked over the surface. If the rheophore is conical, and maintained for some time immovable, it produces a sensation similar to that caused by a hot nail penetrating the skin; hence it is termed the *electric nail*.

(iii.) *Metallic Threads*.—A wire brush may be substituted for one of the ordinary rheophores and moved over the skin, constituting *electric cauterisation*; used to strike it lightly, constituting what is called *electric fustigation*; or held in contact with it, forming an *electric moxa*.

(b) *Neuro-Muscular Faradisation*.—When it is desired to reach the deeper tissues through the skin, it must be remembered that the latter is a bad conductor of electricity, and, consequently, the rheophores should consist of well moistened sponges contained in cylinders, or metallic



discs, covered with wet leather ; and, in addition, the skin itself should be thoroughly moistened with a mixture of warm water and common salt, which increases its electric conductivity. When the muscles are excited to contraction through the skin, the method is called the *percutaneous application*. The current may, however, be passed directly into the muscle which it is desired to contract, by passing needles into its substance ; and this method is called *electro-puncture* or *electric acupuncture*. Electro-puncture was introduced into practice upwards of fifty years ago by Sarlandière, who thrust insulated needles through the skin into the muscles ; but the powerful opposition of Duchenne, along with the brilliant results he himself obtained by the percutaneous application of the current, combined to render the practice of electro-puncture almost entirely obsolete. A few years ago, Dr. Morgan\* introduced the practice of electric acupuncture into the Royal Infirmary, Manchester, being at the time unconscious that it was a revival of an old practice, and no one can have watched, from day to day, the satisfactory results obtained by him without being convinced that this method often offers numerous advantages over percutaneous application. Dr. Morgan at first used insulated needles, but he soon discarded them for needles which are not insulated. "The needles I now use," he says, "are as fine as can be manufactured ; they vary in length from two to four inches, and have a metallic knot, the size of a small pea, attached to one end." Several of these needles are thrust into the muscle which it is desired to excite to contraction, and while one rheophore (generally the anode), with moistened sponge, is placed on an indifferent part of the body, the knobs of the needles are successively touched with the metallic part of the other rheophore, and an instantaneous upward or downward movement of the needle indicates when contraction occurs. The muscles may be excited to contract by passing the faradic current through their substance, either percutaneously or by electro-puncture, or by placing the exciting rheophore over the nerve trunk or branch which supplies the muscle. The former method of application is called *direct muscular faradisation*, and the latter *indirect muscular faradisation*.

(i.) *Direct Muscular Faradisation*.—For the direct electrification of muscles, which present a large surface, like those of the trunk, it is most convenient to use moistened sponges, contained in cylinders ; but when the muscles are small, as those of the face and the interossei, metallic rheophores, with rounded or conical points and covered with wet wash-leather, should be employed. In order to electrify a muscle completely, the rheophores should be applied over its fleshy body, and they should either cover its whole surface or be applied in succession to all points of its surface. When the body of the muscle is very thick, a strong current must be employed, otherwise the superficial layers alone are excited. The points to which the rheophores must be applied, in order to stimulate directly any particular muscle, can only be known from a full acquaintance

\* *The Lancet*, vol. ii., 1879, pp. 454 and 499.

of the anatomy of the muscular system. Sufficient information for the practical use of direct electrification will be found in the special part of this work; and for any further information we must refer the reader to ordinary anatomical works.

(ii.) *Indirect Muscular Faradisation.*—In order to obtain contraction of a muscle, or a group of muscles, by passing a current through the nerve which supplies it, a broad conductor, or a wet sponge contained in a cylinder, should be placed upon an indifferent part of the body, as the sternum, while a fine pointed conductor, such as a conical rheophore, is applied over the most superficial part of the nerve it is desired to excite. To ensure success a minute knowledge of the anatomy of the muscles and nerves, and of their relations to the surface of the body, is essential. It must be remembered, however, that the course and distribution of the nerves are liable to considerable variations, so that the points suitable for excitation—or the *motor points*, as they are called—can only be pointed out approximately. It is, therefore, necessary in practice to make a few trials before the true motor point for a particular group of muscles is ascertained. The motor points for each nerve, trunk, and muscular branch have been carefully determined by Ziemssen, and his figures, copied by permission from Dr. Tibbit's book, will be found in the special part of the work. By the aid of these figures the student can approximately determine the motor points for each nerve and muscle accessible to electrification.

(c) *Faradisation of Internal Organs and Special Senses.*—The faradic current may be applied directly, by means of suitable conductors, to a great many of the internal organs, such as the rectum, bladder, uterus, male genital organs, larynx, œsophagus, and stomach; but for further information upon this subject we must refer the reader to special works on electric therapeutics. The faradic current is but rarely employed in the treatment of the affections of special sense, its use being superseded by that of the galvanic current.

(III.) *Galvanism.*—*The Galvanic, Voltaic, Dynamic, or Constant Current*, as it is variously called, is the electricity of chemical action discovered by Volta and Galvani. The current of electricity passes from the *positive pole or anode to the negative pole or cathode*, and for medical purposes it should be both continuous and constant, inasmuch as any interruptions or considerable variations in the power of the current would alter its character, and render its applications for several purposes both untrustworthy and dangerous. The *intensity* of the current depends on the number of cells employed, and its *quantity* on the size of the elements. The currents used in medicine are of low *tension* and considerable quantity.



### § 172. *Methods of Application.*

All that has already been said with respect to percutaneous faradisation and faradisation by acupuncture, as well as to direct and indirect muscular faradisation, applies equally to the application of the galvanic current. It is unnecessary, therefore, to repeat what has been said with regard to the necessity of having both the rheophores and skin well moistened with warm water and salt when the current is applied percutaneously, or to the choice of the motor points both in direct and indirect muscular galvanisation. Owing to the low tension of the galvanic current its application by acupuncture is of even more importance and value than a similar application of the faradic current, the latter, from its higher tension, being able to penetrate deeper through the tissues.

(1) *Stable Application.*—According to this method the rheophores are maintained immovable on the skin, or the affected extremities are placed in tepid salt water, with which the conducting wires of the battery are in contact.

(2) *Labile Application.*—In this method the cathode is made to glide over the skin, in the direction of the nerves and muscles to be acted on.

(3) *Interrupted Currents and Voltaic Alternatives.*—The constant current acts as a stimulus to both nerves and muscles at the moment of making and breaking contact; hence if it is desired to produce muscular contraction the current is interrupted by suddenly removing one of the rheophores, and reformed by the rheophore being suddenly applied again. When by the aid of a commutator the current is not only interrupted, but also suddenly passed in the opposite direction—a descending current, for instance, being suddenly changed for an ascending current—the method has been called by Remak, who introduced the practice, *voltaic alternatives*. Sudden reversal is a much more powerful stimulant both to nerve and muscle than simple interruption of the current.

(4) *The Direction and Polar Methods.*—In the direction method one rheophore is placed over the plexus, and the other over the trunk of the nerve. When the anode is centrally placed the current is called a *descending current*, and when the cathode occupies that position the current is called an *ascending current*. In the *polar method* one rheophore is placed on an indifferent part of the body, such as the sternum, and the other is placed over the nerve which it is desired to stimulate. It was at one time supposed that these two methods of application involved two different principles of action, but the effects produced can be resolved into the same principles in both cases. When the cathode is used as the exciting pole in the polar method the same kind and degree of contraction is obtained as when the cathode is peripherally placed in the direction method; and, conversely, when the anode is used as the exciting pole in the polar method the reactions obtained are the same as those produced when the anode is peripherally placed in the direction method.

(5) *General Galvanisation* :—

(a) *Centralised Galvanisation.*—Under this name Drs. Beard and

Rockwell have recommended a method of applying the galvanic current with the view of bringing the brain, spinal cord, sympathetic system, and the pneumogastric nerves under its influence. The cathode is placed over the epigastrium; while the anode is passed over the forehead and top of the head, along the inner border of the sterno-mastoid muscle, from the mastoid fossa to the sternum, and at the nape of the neck and down the entire length of the spine. The great aim in centralised galvanisation is to influence the central nervous system, while the aim of general faradisation is to influence the muscular system.

(b) *Electric Bath*.—There are several forms of electric bath, but the simplest is that in which one pole of the battery is connected with the water in which the body is immersed; while the patient grasps in his hands a copper bar covered with wet flannel, and in connection with the second pole of the battery.

(6) *Localised Galvanisation*.—The methods of local application of the galvanic current admit of the same subdivisions as those of the faradic current, namely (a) cutaneous galvanisation, (b) neuro-muscular galvanisation, and (c) galvanisation of the special senses and internal organs.

(a) *Cutaneous Galvanisation*.—In applying the galvanic current to the skin the same precautions must be adopted with respect to the use of dry rheophores as were mentioned in the case of the faradic current. The local application of the galvanic current to the skin has been found useful in the treatment of some skin diseases, as eczema and herpes. Its use in removing small nævi, and, indeed, its surgical uses generally, as well as the applications of electrolysis for the cure of aneurism, will be passed over here entirely as not being part of our subject. In the treatment of peripheral neuralgia one or both poles are placed over the track of the cutaneous sensory nerve, and it is particularly advantageous to place one of the poles in succession over the *points douloureux*. Directions for the selection of these tender spots will be given in the special part of the work.

(b) *Neuro-muscular Galvanisation*.—The law of muscular contraction, as well as the applications of electricity in aiding the diagnosis of the various forms of paralysis, has already been described, and need not be repeated here. Direct galvanisation is found very useful in the treatment of paralysed muscles which manifest the reaction of degeneration. In such cases anodal opening forms the most powerful stimulus to the muscles, and the cathode may be placed on some indifferent point of the body. When the catalytic action of the current is desired a stable or labile application of the current should be employed, and it does not appear to affect the result obtained whether the ascending or descending current be employed.

(c) *Galvanisation of the Special Senses and Internal Organs*.—In galvanisation of the organs of special sense one moistened rheophore is placed upon the face and the second on the organ requiring treatment. In galvanisation of the retina the second moistened rheophore is placed over the closed eye. The retina is peculiarly sensitive to the galvanic current, and



flashes of light are observed from its stimulation when the current is passed through any part of the head and face. In galvanisation of the auditory nerve the external meatus is partly filled with tepid water, and a metallic rheophore, insulated by ivory or vulcanite except at the extremity, is placed in it so as to dip into the water. Galvanisation of the olfactory nerve is effected by moving a metallic sound, insulated except at its extremity, over all the points of the nasal mucous membrane. In applying the current to the tongue the rheophore is moved over the base and borders of the tongue, while the second is placed over the back of the neck.

(d) *Galvanisation of the Brain* may be effected by placing an electrode on each mastoid process, or each temple, or the frontal and occipital protuberances. The sitting should not exceed thirty seconds, and the current should be instantly stopped on the occurrence of the least giddiness.

(e) *Galvanisation of the Sympathetic*.—Benedikt advises galvanisation of the cervical ganglia of the sympathetic for the relief of symptoms of intercranial origin. One electrode may be deeply pressed into the auriculo-maxillary fossæ, and the other with a good-sized sponge applied over the sixth or seventh cervical vertebra, or to the manubrium sterni, close to the border of the sterno-mastoid. The duration should be from one to three or four minutes, and with ten to twenty cells.

(f) *Galvanisation of the Spinal Cord*.—Either the stabile or labile application of the current may be employed in the treatment of the spinal cord. In the former method either pole may be placed on the nape of the neck, while the other is placed over the lumbar region or on a nerve or muscle. When only a small portion of the spinal cord requires treatment the poles must be placed so as to include the diseased portion in the circuit. In the labile application the anode is placed on the nape of the neck or above the limit of the lesion, and the cathode is moved up and down by the sides of the vertebræ, about forty times at each sitting. As the current must penetrate deeply before reaching the spinal cord a strong current must be used, and large moist electrodes, presenting not less than four square inches of surface, should be employed so as to diminish the pain on the surface. Galvanisation of the internal organs may be applied by methods similar to those adopted for the application of the faradic current.

§ 173. *Uses of Electricity*.—The uses of electricity, in the treatment of various nervous diseases, will be described in the special part of this work ; but it will be useful to mention here one or two general principles which underlie all the medical applications of this physical agent. The faradic current is a powerful stimulant to both nerves and muscles, and when a simple stimulant effect is alone desired, this current is, as a rule, more applicable than the galvanic current. The faradic

current is, therefore, a valuable agent in the treatment of all diseases of the nervous system in which the irritability is depressed, no matter whether this declare itself by sensory or motor paralysis. It may, therefore, be used as a cutaneous stimulant, either to act on the skin directly in cases of anæsthesia, or to act on remote organs in a reflex manner; or it may be used as a powerful neuro-muscular stimulant in the various forms of paralysis.

*The Galvanic Current* acts as a stimulant to nerves and muscles both on making and breaking contact, and in addition produces a profound alteration of nutrition during the time the current is interruptedly passing through an organ. The latter action has been called by Remak its catalytic action, and it is to it probably that the constant current owes the many advantages it possesses over the faradic current in the treatment of many of the diseases of the nervous system. The stimulant action of the constant current is, however, very important both as a means of diagnosis and in the treatment of paralysis. We have seen that while paralysed muscles manifest the reaction of degeneration, they are more sensitive to the action of the galvanic than of the faradic current, and in these cases the constant current should be selected to stimulate the nutrition of the affected muscles. With this exception, however, the faradic is a more powerful agent in the direct treatment of paralysed muscles than the constant current. But the catalytic action of the constant current renders it an exceedingly valuable agent in modifying the nutrition of the nerves and trunks of the central organs of the nervous system, and it may even be used in the treatment of cases where the irritability of portions of the nervous system is increased, and to which, therefore, the faradic current is wholly inapplicable. As examples of the numerous applications of the constant current, may be mentioned its employment for the relief of pain in neuralgia, and the assuaging of various forms of spasm, not to speak of its numerous applications both locally and generally in the treatment of chronic diseases of the brain, spinal cord, special senses, and viscera.



§ 174. (4) *To Allay or Remove Serious Symptoms.*

The fourth indication of treatment is to allay or remove serious symptoms, and of these the distressing symptom of *pain* is by far the most prominent and important. Of all the internal remedies which can be used for alleviating pain opium and its various preparations are by far the most important. This drug, however, appears to increase the irritability of the spinal cord, and it must be administered with caution where pain is associated with reflex spasm. Bromide of potassium, chloride of ammonia, chloral, croton chloral, quinine, and various other remedies may at times be used to allay pain, especially the pain of neuralgia, and some of these remedies ought to be used in preference to opium in suitable cases. The constant current is also a valuable agent for assuaging pain, and where it succeeds it should be preferred to all other remedies, inasmuch as its use is not attended with any evil after consequences.

§ 175. *Cold*, continuously applied, has been used as a palliative for the removal of pain in neuralgia; while, at other times, the local application of warmth affords relief, probably by producing relaxation of the tissues in which the nerve fibres are embedded.

§ 176. *Aconite and Veratrine*, applied externally in the form of ointment or liniment, have also been found efficacious for the removal of pain. The liniment or tincture of aconite may be painted over the painful nerve; but a more effectual method is to rub in an ointment containing a grain of aconitine to the drachm of lard, twice a day, so as to maintain complete numbness of the part for two or three consecutive days. Veratrine and atropine ointment may be used in the same way; but the pharmacopœia ointment of the former is too strong.

*Continuous* pressure over a nerve has been employed to arrest its conduction. This treatment has been found useful in allaying the pain of neuralgia and in arresting motor spasm.

§ 177. *Surgical Operations*.—When a nerve is simply divided the operation is called *neurotomy*; but when a portion is dissected out it is called *neurectomy*. Both these operations have been successfully used for the removal of pain or spasm.

Interruption of the continuity of the nerve brings immediate relief, but the pain or spasm is apt to recur when the divided ends of the nerve reunite. Division of a mixed nerve causes paralysis of the muscles supplied by it, so the division or resection of a mixed nerve should only be undertaken under the most pressing need.

§ 178. *Stretching of the affected nerve* is an operation which was first proposed by Nussbaum for the cure of various forms of peripheric neuroses, and its employment has so far been attended with brilliant success. It has not only been found beneficial for the removal of pain and spasm, but it appears to be of great use in the treatment of tetanus and probably other centric affections. The treatment of various other distressing symptoms, such as cystitis, incontinence of urine, painful priapism, and bed-sores, which are apt to supervene in the course of centric diseases of the nervous system, will be described in the special part of the work.



**BOOK II.**

**SPECIAL PATHOLOGY OF THE NERVOUS SYSTEM.**





## PART I.—DISEASES OF THE PERIPHERAL NERVES.

### CHAPTER I.

#### ANATOMICAL INTRODUCTION.

EACH spinal nerve arises by two roots—an anterior which contains the efferent, and a posterior which contains the afferent fibres (Fig. 11, *a a* and *p*). The posterior or sensory root possesses a ganglion the functions of which are not accurately ascertained, although it is known to contain the trophic centre of the afferent fibres. The anterior roots are sensitive, but that they derive their sensibility from the posterior roots is shown by the fact that all sensibility ceases on the latter being divided. The sensibility manifested by the anterior roots has been called “recurrent sensibility,” because it is due to the bending back of sensory fibres along efferent channels.

§ 179. Recurrent sensibility is illustrated by the following diagram :—

FIG. 18.



FIG. 18 (From Hermann's "Physiology").—P R and A are respectively the posterior and anterior roots. The fibres of the posterior root are seen to terminate in the cutaneous surface C; while the fibres of the anterior root terminate in a muscle M; R represents a loop of sensory fibres passing from the posterior roots and bending backward to join A, the anterior root, and furnishing it with recurrent sensibility.

§ 180. *Structure of Nerves*.—The trunks of the spinal nerves consist of:—

(1) Nerve fibres arranged longitudinally to form a bundle (*Fig. 19, N*). The fibres within a bundle are separated from each other by numerous connective tissue fibres, and flattened connective tissue cells, which together constitute the *endoneurium*.

(2) Each bundle is surrounded by a special sheath of connective tissue, called the *perineurium* (*Fig. 19, P*). This sheath possesses a lamellar structure; the lamellæ consist of bundles of fibrous connective tissue, and between them flattened connective tissue cells are observed, lying in spaces which constitute more or less continuous lymph channels. The lymph spaces of the endoneurium have been injected by Axel Key and Retzius in connection with the lymph spaces of the perineurium.

(3) The bundles of nerve fibres are arranged longitudinally, and held together in a common framework—the *epineurium*

FIG. 19.

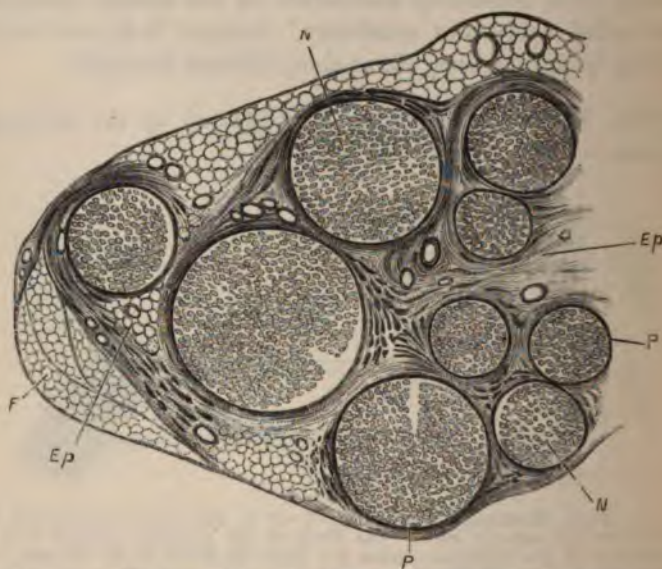


FIG. 19 (From Klein's "Atlas of Histology"). *Transverse Section of a Nerve*.—N, N, Nerve fibres; P, Perineurium; EP, Epineurium; F, Fat cells.



(Fig. 19, EP). This framework is composed of bundles of fibrous connective tissue, arranged as larger or smaller trabeculæ, which cross each other, and thus form a more or less dense plexus. Ordinary flattened and branched connective tissue cells may be observed between the connective tissue bundles; while fat cells, a plexus of lymphatics, and the blood-vessels which supply the nerve trunk, are embedded in the substance of the *epineurium*.

### § 181. *The Deep Origin and Surface Attachments of the Cranial Nerves.*

The first or olfactory nerve or tract must be regarded as a constituent part of the cerebrum and not as a true nerve. It is attached to the under surface of the frontal lobe in front of the anterior perforated space, by means of three roots. The *external or long* root passes outwards and backwards towards the posterior border of the Sylvian fissure where it disappears. The *middle or grey* root consists of grey matter on the surface, which is prolonged from that of the adjacent part of the frontal lobe and the anterior perforated space (Quain). The *internal or short* root is composed of white fibres which may be traced from the inner and posterior part of the frontal lobe. The outer root has been traced to the island of Reil, the optic thalamus, and to a nucleus in the substance of the temporo-sphenoidal lobe in front of the anterior extremity of the hippocampus. It has been supposed that the fibres of the inner root are connected with the anterior extremity of the gyrus fornicatus, or cross over to the opposite hemisphere. The fibres of the middle root are said by some to join those of the inner root, by others to be connected with the corpus striatum (Quain).

The *Optic Tract* arises from the *stratum zonale* and posterior part of the optic thalamus (Pulvinar, Fig. 20, Pv), the internal and external geniculate bodies (Fig. 20, Cgm), and the anterior of the corpora quadrigemina. Additional fibres are derived from the lamina cinerea, and from the tuber cinereum. Some of the fibres, whose functions are probably of reflex character, may be traced to a nucleus in the cerebral peduncles (Stilling). The cortical centre of vision is situated, according to Ferrier, in the angular gyrus; but the researches of Munk appear to show that the visual centre must be placed further back in the occipital lobe. The conducting paths which connect the basal origin of the nerve (the thalamus and geniculate bodies) with the cortical centre pass probably through the posterior part of the internal capsule and corona radiata to reach the convolutions of the occipital lobe.

Immediately after its formation at the external geniculate body the optic tract forms a flattened band, which winds round the cerebral peduncle and tuber cinereum and passes under the lamina cinerea, where

it unites with its fellow of the opposite side to form the optic *commissure* or *chiasma*.

*Chiasma*.—The crossing of the fibres in the chiasma appears to vary considerably in different animals. In the dog, for instance, the crossing is of a somewhat complicated character. Pathological records, however, appear to show that in man the fibres of the optic tract of one side pass to the outer or temporal half of the retina on the same side, and the inner or nasal half of the retina on the opposite side, these being the parts of the retina which are associated in their functions.

*Blood-vessels*.—The optic tract receives at its origin twigs from the arteries distributed to the corpora quadrigemina and the choroid plexus. As it passes under the cerebral peduncle to the chiasma it is covered by the pia mater, from which it derives its vascular supply. The optic nerve receives its arterial supply from the vessels of the tract, and receives additional branches from the posterior or short ciliary arteries.

The *Arteria Centralis Retinae* is derived from the trunk of the ophthalmic artery, or from one of its ciliary or muscular branches, and enters the

FIG. 20.

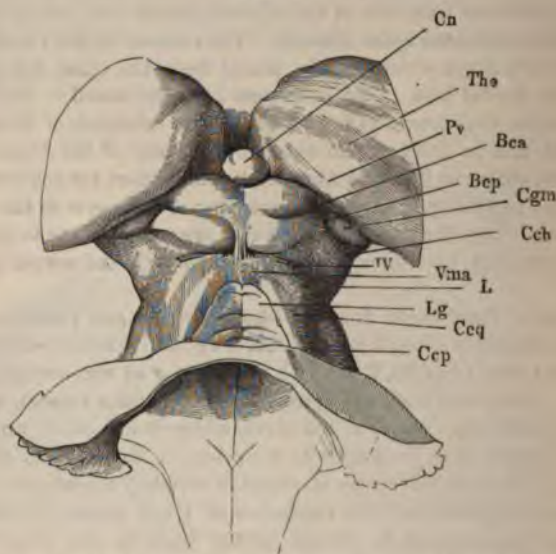


FIG. 20 (From Henle's "Anatomie").—The Corpora Quadrigemina viewed from above after the Cerebellum has been removed. Ccp, Superior Peduncle of the Cerebellum; Ccq, Peduncle of the Cerebellum to the Corpora Quadrigemina; Lg, the Valve of Vieussens; Vma, Velum medulare ant.; Ccb, Crus Cerebri; Cgm, Internal Geniculate Body. The External Geniculate Body is situated close to the outer border of the internal one, but is concealed by the Thalamus. Bca, Bcp, Anterior, and Posterior Brachia connecting the corpora quadrigemina with the Pulvinar; Pv, Pulvinar; Tho, Thalamus Opticus; Cn, Pineal Gland; IV, The Fourth or Trochlear Nerve.



nerve about three-eighths of an inch from the sclerotica. Its main branches are distributed to the retina, although small branches communicate with the nutrient vessels of the optic nerve.

The third and fourth nerves (*oculo-motorius* and *trochlearis*) arise from a grey nucleus, common to both, beneath the floor of the aqueduct of Sylvius. This nucleus communicates with the corpora quadrigemina, and through the crus with the lenticular nucleus. The fourth has also an additional origin from the locus cœruleus.

The third, or oculo-motorius, nerve passes downwards and forwards, and pierces the under and inner surface of the crus cerebri in the interpeduncular space, close to the upper margin of the pons. From this point the nerve passes forwards and outwards, and, after piercing the inner layer of the dura mater, close to the posterior clinoid process, it reaches the external wall of the cavernous sinus, and proceeds forward towards the sphenoidal fissure.

The fourth nerve turns upwards and describes a semicircle around the aqueduct of Sylvius. It then pierces the roof of the aqueduct, and, after decussating with its fellow, crosses to the opposite side, and pierces the crus at its superior and external border (*Fig. 20, IV*). The nerve then passes forwards along the free border of the tentorium to reach the external wall of the cavernous sinus, along which it proceeds to reach the sphenoidal fissure.

The fifth or trifacial nerve arises like a spinal nerve by two roots, the one motor and the other sensory. The latter is like the posterior root of a spinal nerve, inasmuch as it passes through a ganglion—the Gasserian ganglion.

The fibres of the trigeminal nerve take their origin from several nuclei: (*a*) The trigeminal nucleus, which is analogous to the posterior cornu of the grey substance of the cord, and, like the latter, contains only small ganglionic cells. It is situated on a level with the

FIG. 21.



FIG. 21 (From Landois' "Physiologie").—Schema of the semi-decussation of the fibres of the Optic Commissure; *b a*, Left Optic Tract, the fibres of which are distributed to the left halves of both retinae; *b' a'*, the Right Tract, with its fibres supplying the right halves of both retinae.

point of exit of the nerve from the pons and towards the outer part of the floor of the fourth ventricle. (b) An ascending root, derived from the posterior column of the cord from a portion at least as low down as the middle of the neck. The fibres spring from the grey substance of the posterior cornu, and ascend in the white posterior columns. At the side of the medulla the fibres run very superficially, constituting the tubercle of Rolando. (c) *Descending Roots* (1) from the large-celled motor root in the neighbourhood of the corpora quadrigemina. The connections of this root with the ganglia of the brain are not known; (2) from a collection of large vesicular cells (similar to the cells of the spinal ganglia) at the side of the aqueduct of Sylvius; and (3)

FIG. 22.

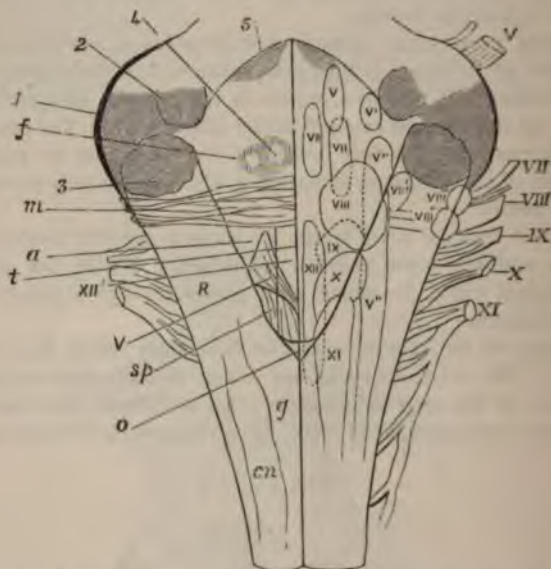


FIG. 22 (after Erb). *View of the Posterior Surface of the Medulla*, the roof of the fourth ventricle being removed to show the rhomboid sinus clearly. —The left half of the figure represents:—Cn, funiculus cuneatus, and g, funiculus gracilis; O, obex; sp, nucleus of the spinal accessory; p, nucleus of pneumogastric, p+sp, ala cinerea; R, restiform body; XII, nucleus of the hypoglossal; t, funiculus teres; a, nucleus of the acoustic; m, striae medullares; s, inferior acoustic nucleus; 1, 2, and 3, middle, superior, and inferior cerebellar peduncles respectively; f, fovea anterior; 4, eminentia teres (genu nervi facialis); 5, locus coeruleus. The right half of the figure represents the nerve nuclei diagrammatically; V, motor trigeminal nucleus; V', median, and V'', inferior sensory trigeminal nuclei; VI, nucleus of abducens; VII, facial nucleus; VIII, posterior median acoustic nucleus; VIII', anterior median, VIII'' posterior lateral, VIII''' anterior lateral acoustic nuclei; IX, glosso-pharyngeal nucleus; X, XI, and XII, nuclei of vagus, spinal accessory, and hypoglossal nerves respectively. The Roman numerals at the side of the figure, from V to XII, represent the corresponding nerve roots.



from the locus cœruleus, which lies beneath the *substantia ferruginea* in the upper part of the floor of the fourth ventricle. (d) Fibres from the cerebellum, running in the *crura cerebelli* and *corpora quadrigemina*.

The nerve issues from the side of the pons considerably nearer the upper than the lower border. The smaller root is at first concealed by the larger, and is placed a little higher up. Both roots are directed forwards side by side to the middle fossa of the skull, through a recess in the *dura mater* on the summit of the petrous part of the temporal bone, at the apex of which the Gasserian ganglion is lodged in a depression, and receives on its inner side filaments from the carotid plexus of the sympathetic nerve. The smaller root passes inside and beneath the ganglion without communicating with it, and outside the skull it joins the lowest of the three trunks which issue from the ganglion.

*The Sixth Nerve (abducens)* arises from a large-celled nucleus at the bottom of the groove in the floor of the fourth ventricle at the junction of the medulla oblongata and pons near the *fasciculus teres*. This nucleus communicates with the nucleus of the third nerve of the opposite side (Duval). After the emergence of the sixth nerve from the medulla oblongata it passes forwards and outwards over the posterior and smooth quadrilateral surface of the sphenoid bone (*dorsum sellæ*), and enters the cavernous sinus, between the internal carotid artery and the fourth nerve. It then passes forwards on the floor of the sinus, close to the outer side of the carotid artery, and enters the orbit through the sphenoidal fissure between the heads of the external rectus muscle.

*The Seventh or Facial Nerve* is derived from a nucleus similar to that of the sixth, but situated somewhat lower, and more in the substance of the medulla oblongata. It was supposed that the nerve received a number of fibres from the nucleus of the sixth, but the observations of Gowers appear to have disposed of this statement. A descending set of fibres comes from the lenticular nucleus of the opposite side of the body. The facial nerves appear on each side of the medulla at the inferior margin of the pons. Each nerve emerges in the outer margin of the depression between the olivary body and the diverging restiform body, the auditory nerve lying to its outer side. The nerve passes outwards from its place of origin to enter the internal auditory meatus, and at the bottom of the meatus it enters the aqueduct of Fallopius and follows its windings to the lower surface of the skull.

*The Eighth or Auditory Nerve* receives fibres from four independent nuclei which lie on a level with the broadest portion of the fourth ventricle. (1) The posterior median nucleus (*Fig. 22, VIII*), occupying the whole space included between the *ala cinerea* and the inferior cerebellar peduncle up to the anterior border of the *striæ medullares*. The posterior root of the acoustic takes its chief origin from this nucleus, and passes out partly in superficial fasciculi (*striæ acousticæ*) and partly through the body of the medulla. (2) Anterior median acoustic nucleus (*Fig. 22,*

VIII'), occupying the external angle of the fourth ventricle, about the middle of the inferior cerebellar peduncle. (3) Posterior lateral acoustic nucleus (*Fig. 22, VIII'*) lies in the form of a small grey nodule between the fasciculi of origin of the acoustic nerve at its point of emergence from the medulla. (4) Anterior lateral acoustic nucleus (*Fig. 22, VIII''*). It appears to be a prolongation of the posterior lateral nucleus, and is wedged in between the middle peduncle of the cerebellum and the flocculus.

*The Ninth, or Glosso-pharyngeal Nerve.*—The nucleus of the glosso-pharyngeal nerve is not separated by a distinct boundary from that of the vagus, but the former lies somewhat more superficially than the latter (*Fig. 22, IX*).

*The Tenth Nerve (Par Vagus, Pneumogastric or Vagus).*—The vagus arises from a nucleus situated in the lower half of the floor of the fourth

FIG. 23.



FIG. 23 (after Erb). *Transparent lateral view of the Medulla, showing the relative positions of the most important nuclei; right half of the medulla, seen from the surface of section; the parts that lie closer to this surface are deeper shaded. Diagramatic. Py, pyramidal tract; Py, Kr, decussation of pyramids; O, olivary body; Os, superior olivary body; V, motor, V', middle sensory, V'', inferior sensory nucleus of trigeminus; VI, nucleus of abducens; Cf, genu facialis nervi; VII, nucleus facialis; VIII, posterior median acoustic nucleus; IX, Glosso-pharyngeal nucleus; X, nucleus of vagus; XI, nucleus of the accessorius; XII, hypoglossal nucleus; Kz, nucleus of the funiculus gracilis; Re, trigeminal root; RVII, root of the facialis; RVII, root of the facialis.*



ventricle (*Fig. 23, X*), and from another in the substance of the medulla oblongata, near the olivary body. The nerve emerges from the medulla oblongata, between its lateral column and the restiform body. Its roots, between twelve or fifteen in number, lie beneath, and in a line with the roots of the glosso-pharyngeal nerve. The filaments of the roots are arranged in a flat fasciculus, which is directed outwards, with the glosso-pharyngeal nerve, across the flocculus to the jugular foramen.

*The Eleventh, or Spinal Accessory Nerve*, consists of two branches—an internal, derived from the medulla, which joins the trunk of the pneumogastric, and an external branch derived from the cord, which is distributed to the sterno-mastoid and trapezius muscles. This nerve arises by a series of roots, the upper of which are attached to the side of the medulla below those of the pneumogastric, while the remainder arise from the lateral column of the cord as low down as the sixth or seventh pair of cervical nerves. The upper roots pass inwards to a nucleus which lies on each side, at the back of, and close to the central canal. The spinal roots pass through the lateral column of the cord, and enter the grey substance midway between the anterior and posterior cornua.

*The Twelfth or Hypoglossal Nerve*.—The nucleus of the hypoglossal consists of a column of large branching nerve cells, which lies close to and in front of the central canal, as low as the decussation of the pyramids. When the canal opens into the floor of the fourth ventricle the nucleus comes to the surface, and causes a prominence close to the middle line, a little above the point of the calamus scriptorius.

Bundles of fibres pass forwards from the nucleus, through the inner side of the olivary body, to form a series of fine roots in the furrow between the anterior pyramid of the medulla and the olivary body. The roots are collected into two bundles, which converge to the anterior condyloid foramen of the occipital bone. These bundles perforate the dura mater separately within the foramen, and are joined into one trunk after they have passed through it.

The subjoined diagrams (*Figs. 24 and 25*) show the surface attachments of the cranial nerves, and their course through the skull, so clearly as to render unnecessary a detailed reference to them in the text.

FIG. 24.

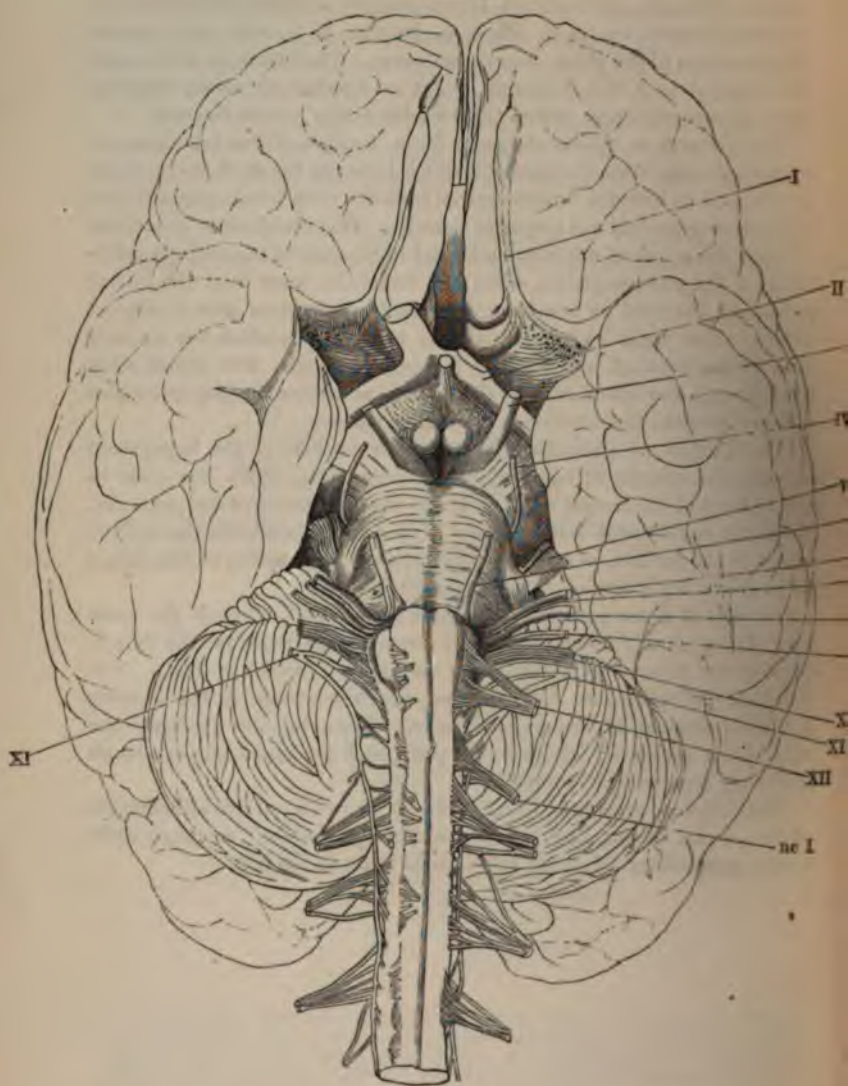


FIG. 24 (From Henlé's "Anatomie"). *The Base of the Brain and adjoining part of the Spinal Cord.*—The Cranial Nerves are represented by the corresponding Roman letters from I to XII. VII', Portio intermedia of the seventh; nc I, First cervical nerve.



FIG. 25.

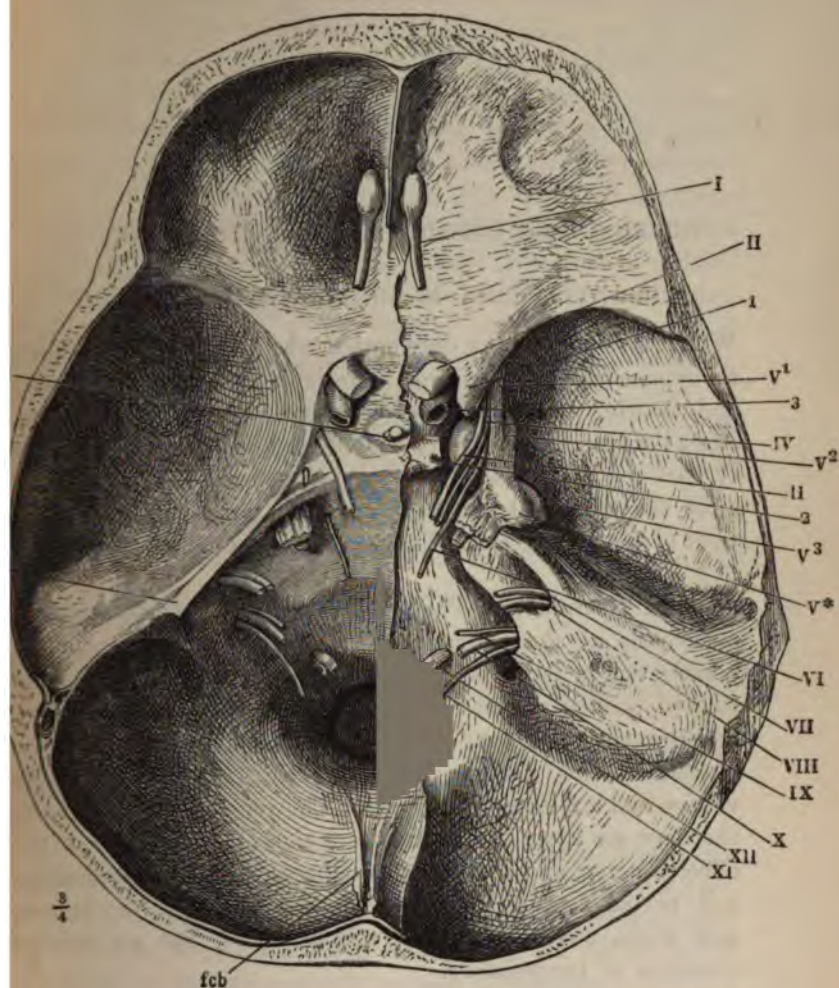


FIG. 25 (From Henle's "Anatomie"). *Internal View of the Base of the Skull, showing the Places of Exit of the Cranial Nerves.*—The Nerves are represented by the corresponding Roman numerals, from I to XII.  $V^1$ ,  $V^2$ ,  $V^3$ , the first, second, and third divisions of the fifth respectively.  $V^*$ , the Gasserian Ganglion. The sensory root is cut short in order to show the motor root of the fifth as it passes under the ganglion. The dura mater is removed on the right side, and the nerves may be followed to the various foramina through which they pass from the skull. The Nerves III, IV,  $V^1$ , and VI may be seen passing over the convexity of the curve, which the internal carotid artery makes in the groove of the sphenoid bone. 1, Anterior clinoid process; 2, Posterior clinoid process; 3, Transverse section of the internal carotid artery. H, Peduncle of the pituitary body; t, anterior body of the divided tentorium; fcb, Falx Cerebelli.

## CHAPTER II.

## GENERAL DISEASES OF THE PERIPHERAL NERVES.

## (I.)—HYPERÆMIA OF THE NERVES—CONGESTION.

WEIR MITCHELL is the only author who has directed attention to hyperæmia of the nerves. He employed artificial freezing and thawing of exposed nerves as a means of investigation. After thawing, the nerves presented more or less extensive rosy or dark-red injections, and appeared somewhat swollen, and after the prolonged application of cold punctiform extravasations of blood were found between the nerve fibres.

§ 182. *Symptoms.*—The symptoms due to freezing are pain, anæsthesia, paralysis, increase of temperature, and augmented perspiration in the region of distribution of the nerve. In the thawed portion of the nerve a very painful sensation is felt, which in the case of the ulnar nerve spreads backwards to the brachial plexus, and, in some instances, may even produce vertigo and a feeling of faintness. The symptoms which follow the thaw are hyperæsthesia in the region of distribution of the affected nerve, and a feeling of numbness, creeping, formication, and partial loss of power, along with a sense of fulness, and slight swelling of the affected part, but no notable elevation of temperature.

## (II.)—INFLAMMATION OF NERVES—NEURITIS.

The frequency with which neuritis occurs is not yet definitely settled, but there can be no doubt that it occurs much more frequently than was at one time supposed, although it is very probable that some authors, like Remak and Benedict, attributed too great importance to inflammation of nerves.



*Etiology.*—Wounds of various kinds, contusion, rupture, laceration, and the penetration of foreign bodies, are the best known and most frequent causes of neuritis. Strong compression of a nerve, sudden and violent muscular movements, severe concussions of a nerve from long travelling in a badly-constructed waggon, and various other injuries may also cause neuritis. Exposure to cold, wet feet, and standing in ice cold water, have also been observed to occasion the disease. Neuritis may also result from extension of inflammation from the surrounding tissues and organs. Pleurisy, pleuro-pneumonia, and tuberculosis of the lungs have caused inflammation of the intercostal nerves, as proved by Beau. Acute and chronic rheumatism of the joints, especially of the shoulder joint, not unfrequently lead to neuritis. Caries of bones, inflamed tendinous sheaths, and malignant growths may be mentioned as other causes of the disease. Neuritis frequently becomes developed after acute diseases, such as typhoid fever, the acute exanthemata, and diphtheria; and it is also often caused by many chronic diseases, such as syphilis. Inflammation of the nerves is also met with in lepra anæsthetica.

Neuritis sometimes appears in the absence of any recognisable cause, and then we must speak of its origin as spontaneous and regard it as *idiopathic neuritis*.

*Symptoms.*—The symptoms vary according as the disease is *acute* or *chronic*.

§ 183. *Acute Neuritis* comes on shortly after exposure to one or other of the causes of the disease. It is ushered in by a well-marked feeling of chilliness or by an actual rigor accompanied by headache, sleeplessness, and smart fever. In inflammation of a mixed or sensory nerve the patient experiences severe and almost intolerable pain, occurring in the absence of all external causes. The pain is usually limited to the region supplied by the affected nerve, but when very intense it may radiate to the regions of other nerves of the same plexus, and even into more remote nerve territories. The pain is generally intense, deep-seated, tearing, boring, or burning, and it is described as *almost continuous*. Perfectly free intervals, such as occur in neuralgia, are not observed in neuritis, although the symptoms

are characterised by remissions and paroxysmal exacerbations. The latter are specially apt to occur at night. If the neuritis is circumscribed, paroxysms of neuralgic pain may, according to Nothnagel, be present, separated by free intervals; but the pain at the inflamed spot is continuous. Every movement of the limb and everything that excites the inflammation augments the pain. In excitable patients acute neuritis may produce great agitation of the whole system, slight delirium, and an emotional condition, resembling an attack of hysteria.

Inflamed sensory or mixed nerves are always sensitive to pressure or pinching. The pain caused by pressure shows itself at the inflamed spot, and also radiates out from it both in centripetal and centrifugal directions. The skin over the nerve and in the whole area of its distribution exhibits a marked degree of hyperæsthesia, and this is accompanied by sensations of numbness and formication; and the hyperæsthesia soon gives place to anæsthesia when the exudation begins to compress the nerve fibres.

In some cases motor irritative phenomena, such as twitchings and contractions, are present, but these soon give place to symptoms of paresis or paralysis, which may vary in degree from a slight feeling of fatigue when movements are made to complete paralysis. The muscles supplied by the nerve frequently undergo rapid atrophy. In some cases a red line resembling a superficial lymphanginitis is observed along the course of the inflamed nerve.

§ 184. *Chronic Neuritis*.—Chronic neuritis occurs either as a sequel to an acute attack or arises insidiously with obscure symptoms, gradually or suddenly rising to great intensity. Pain is the earliest and most constant symptom. It varies considerably in character and intensity, being sometimes dull and tensive; at other times of a lancinating or tearing character, and radiating towards the periphery. The pain is continuous, although frequently interrupted by paroxysmal exacerbations, which generally occur at night and prevent sleep. It is increased by every kind of exertion and movement, and by everything which excites the activity of the heart. The patient also complains of numbness, creeping, formication, and of unpleasant pricking sensations, when the skin is touched or struck.



Symptoms of motor irritation, such as tension of the muscles, tremors, sudden contractions, and occasionally violent tonic cramps and persistent contractures, are observed, and these are succeeded by paralytic phenomena.

Anæsthesia, varying in degree from slight blunting to complete loss of all forms of sensibility, is observed in the second stage of the affection.

During the primary or irritative stage of the affection, symptoms of radiation and various reflex phenomena are commonly observed. Reflex cramp is sometimes so violent that the nails of the fingers, for example, may bury themselves in the skin of the hand, from spasm of the flexors. Occasionally neuritis gives rise to general convulsions, hysterical convulsive attacks, and even well-marked tetanus.

Swelling of the affected nerve may take place, so that when it occupies a superficial position it can be felt as a continuously thickened cord. In other cases the thickening occurs at certain intervals, so as to give rise to fusiform or moniliform swellings in its course (*neuritis nodosa*). The nerve is always sensitive to pressure, and pressure made upon one of the swelled portions of nerve occasions excentric sensations, as pain and formication.

With regard to the electric relations, the excitability for both currents is increased in slight cases. If the paralysis is well marked the electrical excitation may remain normal, as in slight cases proceeding from compression; but if the disease has advanced to degeneration of the nerves, the reaction of degeneration is always present. The muscles become greatly atrophied, as a rule, but this is not a necessary symptom of neuritis. Friedreich is inclined to regard all cases of neuralgia, in which muscular atrophy supervenes, as being caused by neuritis.

Trophic disturbances in the skin and nails, swelling and stiffness of the joints, have frequently been observed in neuritis; and herpes zoster is probably dependent upon a similar affection. Epilepsy, as well as traumatic trismus and tetanus, may also be induced by neuritis.

*Duration, Course, and Termination.*—Acute neuritis lasts for a few days or weeks, and either terminates in recovery or passes into the chronic form. Chronic neuritis is of very uncertain, but always of protracted duration, and even the

slighter forms do not recover before the lapse of weeks or months. The traumatic forms are the least complicated when reunion has taken place; the disturbances of sensibility first disappear, motor power then gradually returns, and ultimately the atrophy and any secondary disturbances of nutrition that may be present gradually vanish, and the recovery is complete. Idiopathic forms of neuritis, those dependent on rheumatic causes, or which occur after acute diseases, are less favourable, and may last for months or years, and in some cases never entirely disappear. Persistent neuralgia and anæsthesia, debility, and even complete motor paralysis, not unfrequently result from chronic neuritis; but improvement and recovery may take place even in these cases at a late period of the disease. The course and symptoms of neuritis vary according as the nerves affected are purely sensory, purely motor, or mixed, but the symptoms in each case may be deduced from the general description which has already been given.

Neuritis manifests a constant disposition to propagate itself along the nerve in a centripetal direction, and then to extend to nerves lying at a higher plane and to the spinal cord. Leyden is inclined to refer the myelitis, which occurs after affections of the urinary and other pelvic viscera, to a lumbo-sacral ascending neuritis, extending to the spinal cord; and Duménil has observed wide-spread chronic myelitis result from primary ascending chronic neuritis. Friedreich regards neuritis as the middle term between primary myositis and what he regards as the secondary affection of the spinal cord in progressive muscular atrophy; and he also refers numerous other morbid processes either to ascending or descending neuritis.

#### § 185. *Morbid Anatomy.*

Inflammation of a nerve may begin in the nerve fibres themselves, and it then constitutes a true *neuritis*; or it may begin in the sheath or epineurium, when it constitutes *perineuritis*. But inasmuch as the different layers of the sheath of the nerve are rapidly invaded by the inflammatory process when the nerve fibres are primarily affected, and the nerve fibres are readily implicated when the process begins in the sheath, it is not always easy to distinguish in practice between



*neuritis* and *perineuritis*. In acute neuritis or perineuritis the vessels become enlarged and distended, and the nerve trunk swollen from serous, gelatinous, or fibrinous exudation. As the disease proceeds, the medulla coagulates, and both the inter-annular nuclei of the nerve fibres and the connective tissue nuclei undergo proliferation. During the stage of serous effusion the nerve may be unusually soft, but when the effusion becomes fibrinous the nerve becomes abnormally firm.

The further progress of the case depends upon the acuteness of the process and the length of time which elapses before the inflammatory action is arrested. If the inflammation subsides at an early date the effusion is absorbed before there is any destruction of the nerve fibres, and the healthy condition is readily re-established. If the inflammatory action is very acute and severe both white and red blood corpuscles escape from the vessels, the colour of the nerve becomes yellow or brownish red, its tissue is infiltrated with sanguineous pus, while abscesses may form around its trunk (Mitchell), and the entire structure may become completely disintegrated.

When the process has passed from the acute into the chronic form, or has been chronic from the beginning, the trunk of the nerve becomes irregularly vascular, while it becomes enlarged in some places and atrophied at others. The sheath of the nerve becomes thickened, fibrous, resisting, and frequently adherent to the adjacent tissues. In cases of perineuritis the nerve fibres are compressed by the exudation and disappear after a time, so that the structure of the nerve is supplanted by a band of connective tissue.

#### § 186. *Diagnosis.*

Where pain and paræsthesiæ occur in the area of distribution of a certain nerve, along with symptoms of sensory and motor irritation, followed by paralysis, and when at the same time painful swelling of the nerve can be detected, there can be no doubt that neuritis is present. Chronic neuritis may easily be mistaken for neuralgia; and, indeed, the distinction between the two is by no means clear at any time. If the pain be continuous and the affected nerve tender over a large portion of its track, and not simply over the points of Valleix, and when

indications of sensory and motor paresis are of early occurrence, then the cause is probably neuritis. Neuritis is readily distinguished from muscular *rheumatism* by the seat and extent of the pain, whilst no pain is felt as in the latter affection during contraction of the affected muscles, or when pressure is made over them.

*Thrombosis and embolism* of the larger vessels of the extremities may be recognised by the coincident disturbances of the circulation, and by the œdema and other symptoms.

The diagnosis between neuritis and diseases of the central organs of the nervous system will be described in subsequent sections.

#### § 187. *Prognosis.*

The prognosis is always doubtful, both on account of the long duration of the disease, the secondary paralytic and trophic symptoms which supervene, and the possible transference of the morbid processes to the central nervous system. The prognosis of the acute traumatic form is relatively favourable, and the same may be said of the subacute and chronic forms, which result from injury of the nerves. Idiopathic and rheumatic forms are frequently very obstinate and tedious, and only the slight cases are likely to terminate favourably. The prognosis must depend in every case on the causes and extent of the lesion, and especially upon the individual peculiarities which may be present.

#### § 188. *Treatment.*

The treatment must first be directed to the removal of the cause, and surgical interference is frequently necessary in cases of injury. At other times the treatment must be directed against articular rheumatism, inflammation of tendons, syphilis, and other morbid conditions.

In simple congestion the steady application of ice along the track of the affected nerve, elevation of the part, and absolute rest suffice to arrest the disease.

If *acute neuritis* has been established, energetic antiphlogistic treatment must be adopted—local depletion, application of ice, purgatives, favourable position of the parts, and absolute



rest. Large doses of quinine may be useful, and the subcutaneous injection of morphia and atropia may be necessary to allay pain.

In the *subacute and chronic forms* the use of the antiphlogistic treatment must be less active. Cold may be applied in the form of elongated ice-bottles covering the nerve throughout its whole length; or the wet bandages of Priessnitz may be used, with subsequent employment of cool hip baths (Erb). In the later stages of the affection counter irritants are very useful, and the faradic brush may be applied over as large a surface as possible, or painting with iodine and blistering may be tried.

The *galvanic current* is the great remedy for all forms of chronic neuritis. The best mode of employing it is to keep the anode steadily applied to the affected spot every day for a few minutes; but other modes of application may be required to meet the special circumstances of the case. If the symptoms be severe, absolute rest will be required, assisted by an appropriate position. The patient should be forbidden to take exercise or to perform any kind of work requiring exertion, especially that involving exposure to cold water.

In very obstinate and chronic cases, recourse may be had to energetic counter irritants, such as the moxa and actual cautery. Hot baths, such as those of Wilbad, Gastein, Teplitz, Wiesbaden, mud, and strong saline baths have also been found useful in the treatment of neuritis.

### § 189. *Atrophy of the Nerves.*

Atrophy of the nerves is generally observed as a secondary affection of other abnormal conditions, as wounds, compression, and inflammation of nerves, although primary or idiopathic atrophy of the peripheral nerves does occasionally occur.

In *Idiopathic Atrophy* the nerve is diminished in size and has a grey semi-transparent aspect, there is a moderate hypertrophy of connective tissue, and simple disappearance of the nerve fibrils without fatty degeneration of the medulla.

*Secondary Atrophy.*—The histological changes which accompany secondary atrophy of the nerves have already been described in the chapter on the Trophoneuroses. The atrophied nerve appears to the naked eye as a pale grey, translucent, slender,

flattened band; its sheath is sometimes flaccid, but more frequently thickened from hypertrophy of the connective tissue, and coalesced with the surrounding tissues.

Atrophy of the nerves is observed in anencephalic monsters, spina bifida (Rokitansky), paralytic idiocy, bulbar paralysis, many forms of myelitis, tabes dorsalis, spinal paralysis of children, general paralysis of adults, progressive muscular atrophy, section or compression of nerves, and neuritis. Secondary atrophy of nerves is sometimes caused by destruction or abolition of the functions of peripheral organs. There occurs in these cases a centripetally extending atrophy. The form which occurs in the optic nerves is the best known. Simple atrophy occurs at an early period after extirpation or destruction of the globe of the eye, and after glaucoma; and at a later period the optic nerves become grey and transparent, and present the histological characters of grey degeneration. Many years after an amputation of an extremity has been performed, a centripetally progressive atrophy of the divided nerves has been observed, often extending to the spinal cord, and appearing as a simple attenuation of the nerves, or accompanied by all the signs of neuritic atrophy.

*Symptoms.*—Little is known of idiopathic atrophy of the peripheral nerves, with the exception of the optic nerve, and secondary atrophy is a subsidiary symptom of the primary disease. It may be laid down as a general law that wherever atrophy is developed the function of the nerve is lowered or lost. Atrophy of nerves may be recognised by electrical examination, and in the case of the optic nerve by the ophthalmoscope. When the faradic and galvanic irritability are much below the normal, or altogether lost in cases where the muscles are not completely atrophied, it may be inferred that a condition of degeneration and atrophy of the nerve supplying them exists.

*The prognosis* varies with the nature of the primary disease. If recovery from the conditions causing the atrophy be possible, then the function of the nerve may be restored; but if that be impossible, or if the atrophy has persisted for a long period, the prognosis is unfavourable.

*Treatment.*—The treatment depends essentially upon the



primary disease, and when this can be removed recovery from the atrophy may be expected. Electricity is the best remedy in the treatment of the atrophy itself, and the galvanic current is by far the best method of employing it. Even atrophy of the optic nerve appears to have been much benefited by the use of the galvanic current. In the paralyses which accompany atrophy of a nerve, energetic shampooing of the limbs, warm baths, saline baths, alternate warm and cold douches, friction, either alone or with spirituous and stimulating liniments, may be employed.

(III).—HYPERTROPHY AND NEOPLASTIC FORMATIONS IN THE NERVES.

§ 190. *True and False Neuromata.*

Hypertrophy of the peripheral nerves is so rare as to be regarded a mere curiosity.

The hypertrophy, as a rule, consists of an increase of volume caused by interstitial growth of connective tissue. Occasionally, however, there is true hyperplasia of the nerve tissue, consisting of an increase in the number of nerve fibres, division or splitting of the fibres into a large number of daughter fibres, great thickness of the medullary sheath, and even of the axis-cylinder.

Remarkable thickening and hypertrophy of the nerves have been observed in many instances of elephantiasis, and also in other diseases.

*Neoplastic Formations.*—Growths in nerves may be subdivided into two classes—*true* and *false* neuromata. True neuromata consist wholly or chiefly of true nerve substance; while false neuromata, although seated on the nerves and proceeding from them, are not composed of nerve tissue.

§ 191. *Pathological Anatomy.*

*True Neuroma.*—This growth consists chiefly of nerve fibres, more or less mixed with connective tissue which is sometimes soft, sometimes tough, sometimes vascular, and at other times almost destitute of vessels. Various forms of neuromata have been distinguished, such as fibro-neuroma, glio-neuroma, myxo-neuroma, neuroma teleangiectodes, &c. It is doubtful whether ganglion cells have ever been found in neuromata.

Neuromata occur almost exclusively in spinal nerves, rarely in sympathetic nerves, and still more rarely in one of the cerebral nerves. Their size varies from that of a millet seed to that of the fist.

There are two forms of true neuromata. In one form medullated double contoured fibres are common, and they give to the tumour a medullary white aspect; hence it has been called *neuroma myelinicum* by Virchow. This form is frequently met with after amputations. In the other form, extremely fine non-medullated fibres are present, giving a grey colour to the tumour, and which are blended so as to form a felt-like mass, resembling fibro-myoma of the uterus. This variety has been called, by Virchow, *neuroma amyelinicum*. It was formerly mistaken for fibroma or fibro-sarcoma.

*False Neuroma.*—The main portion of the tumour does not consist of nerve tissue, and fibres proceeding from the affected nerve into the tumour do not appear to be affected. Various tumours belong to the category of false neuromata.

(a) *Fibromata.*—These tumours are composed of connective tissue, enclosing a few nerve fibres. They appear in the form of dense small knots, and the majority of the small tumours in the nerves called *tubercula dolorosa* consist of fibrous tissue, although these small and very painful tubercles may belong to any of the varieties of tumours found in nerves.

(b) *Myxomata.*—These tumours are formed of mucous tissue, and they are frequently met with in nerves. They consist of soft, lobulated, reddish, transparent, gelatinous growths, and the characteristic stellate intercommunicating cells are found on microscopic examination. Cystic formations are sometimes found in the myxomata, leading to the formation of a group termed *neuroma cysticum*.

(c) *Gliomata* have hitherto only been found in the auditory nerve.

(d) *Sarcomata* of various kinds occur in nerves; and transitional forms between fibroma and myxoma are not unfrequently observed.

(e) *Carcinomata* are frequently met with and under various forms. Scirrhus, and medullary cancer are often observed, and melenoid cancer is also not uncommon. Cancer is, as a



rule, secondary ; but occasionally it is primary, and it always causes more or less complete destruction and degeneration of the nerve fibres.

(f) *Syphilitic Gummata* sometimes form in the cerebral nerves at the base of the cranium, and are generally propagated from the membranes of the brain.

(g) *Lepra Nervorum* generally appears as a diffuse, more or less fusiform, swelling of the nerves. It consists of a development of granulation tissue, which is often indistinguishable from that produced in inflammation.

The size of these tumours is extremely variable. Some are not larger than a mustard seed, while others grow to the size of a man's head ; but the majority of them range from between the size of a bean and that of a hen's egg.

The number of the tumours may also be very variable. At times there is only a solitary tumour, while at other times a large number may be present either at a circumscribed spot or distributed over the body. When the tumours are *locally* numerous they may either form a series of knots in the same nerve, or numerous knots in the various branches of one trunk or plexus. At other times the tumours may occur in large numbers, amounting sometimes to several thousands, in all parts of the body, chiefly in the spinal and occasionally also in the cerebral nerves.

The relations of these various tumours to the nerves may be very diverse. At times the new formation is on one side of the nerve, so that the latter seems to run over its surface ; at other times it occupies the centre of the nerve ; while in still other cases the nerve runs directly into the tumour, the fibres breaking up into a kind of brush or pencil. In true neuromata either the whole or a portion of the fibres of the nerve participate in the new formation ; false neuromata proceed for the most part from the neurilemma, and the nerve fibres may remain more or less intact, or they may be compressed and completely destroyed.

§ 192. *Etiology*.—Some individuals appear to have a certain predisposition towards the formation of neuromata, and phthisical and scrofulous persons seem to be specially liable to their formation. Isolated neuromata are more common in women, while multiple neuromata occur almost exclusively in men. They occur at all ages, and are often congenital.

The best known of the direct causes are blows, pressure, penetration, and retention of foreign bodies, and various other injuries. Neuromata are also frequently found in cicatrices when nerves are divided or injured, and rounded and elongated

swellings of the nerves are frequently found in the cicatrices of the stumps after amputation.

Chronic neuritis may also be the starting point of neuromata; and syphilis, lepra, and elephantiasis may lead to the formation of tumours in nerves, but in a large number of cases no definite cause can be traced.

§ 193. *Symptoms.*—The symptoms of neuromata are variable. Many tumours cause no symptoms throughout, while others occasion intense and persistent suffering. Isolated neuromata generally give rise to severe and incurable neuralgia, hence they have been called *tubercula dolorosa*. The pain may be tearing, lancinating, aching, boring, or burning; it is almost always remittent, or completely intermittent; but when the paroxysm comes on, the pain gradually increases in intensity, and radiates from certain points towards the periphery. The pain is increased by cold and damp weather, by pressure, or the slightest movement of the affected limb; and in women, frequently by the return of the menses, or by pregnancy. It may often be made to disappear temporarily by firm pressure on the nerve above the tumour. The pain is generally more severe in small tumours seated on peripheral cutaneous branches than in larger tumours of deeper seated nerve trunks.

In addition to pain, a feeling of numbness and formication, and a sensation of heat and cold are often felt in the area of the peripheral distribution of the affected nerve.

*Motor disturbances* are rare, but sometimes occur in the form of tremors, spasms, and contractures, and these may ultimately give place to complete paralysis. Anæsthesia may also be present in various degrees, and not unfrequently appears in the form of *anæsthesia dolorosa*, especially in cancer. In very excitable persons diffused pain may occur in the head and spine; and occasionally epileptoid or tetanoid convulsions may occur during the paroxysms of pain.

The further course of the disease varies in different instances. The symptoms may become so intense that the patient experiences the most frightful suffering, whilst the constant sleeplessness and the secondary disturbance may lead to a high degree of cachexia, exhaustion, and even death.



At other times the symptoms may remit and ultimately cease, and in a few cases the tumour disappears.

In other cases symptoms of paralysis supervene, and tumour of the cauda equina causes paraplegia and various trophic disturbances.

True neuromata may remain stationary for many years without producing any deleterious effect.

*Multiple neuromata* present in many cases scarcely any symptoms, and they are often only discovered accidentally during life or at the autopsy. They may, however, cause various disturbances by their mechanical action, and when the sympathetic nerves are implicated they may induce general debility and anæmia and various other anomalous symptoms.

The *diagnosis* of neuromata is founded almost exclusively on the presence, on certain nerves, of round or oval tumours, of various sizes, which are movable from side to side, but not in the direction of the length of the nerves.

*Prognosis.*—True neuroma is always a local and benign affection; but it may occasionally return once or several times after extirpation. The prognosis of spurious neuromata depends upon the nature of the tumour, and is the same as when they develop in other tissues.

The prognosis is bad when the affection is accompanied by severe neuralgic pains, and serious trophic and vaso-motor disturbances, and when extirpation is impracticable.

§ 194. *Treatment.*—The only successful treatment of neuromata is afforded by the extirpation or destruction of the tumour. Extirpation succeeds best when the growth can be removed and the nerve left intact. When extirpation is impossible, destruction of the tumour may be undertaken by caustics or electrolysis, but this method is not very successful. When the tumour cannot be removed, palliative treatment by means of narcotics and other means must be employed to alleviate the sufferings of the patient.

## CHAPTER III.

## DISEASES OF THE NERVES OF SPECIAL SENSE.

## (I.)—DISEASES OF THE OLFACTORY NERVE.

THE sense of smell is excited by the contact of odoriferous particles with the mucous membrane of the nose, or rather with that part of it to which the olfactory nerves are distributed. Two delicate, scroll-like bones, called *turbinated* bones, are attached to the lateral walls of each nasal cavity, and divide the latter into three chambers, the one being above the other. The uppermost two of these constitute the true olfactory chambers, whilst the lowest passage is merely used for respiratory purposes. Peculiar rod-shaped cells, described by Max Schultze, which are attached to the ramifications of the olfactory nerves in the mucous membrane of the nose, appear to be the peripheral end-organs of smell.

When odoriferous particles are present in the inspired air passing through the lower nasal chambers, they diffuse into the upper chambers, and, on coming in contact with the olfactory epithelium, originate the impulses which cause the sensation of smell. The smell induced by simple diffusion of odoriferous particles is, however, usually very imperfect; but a more complete contact of those particles with the olfactory mucous membrane is obtained by the forcible nasal inspiration called *sniffing*. The mucous membrane of the nose not only possesses specific olfactory sensation, but also sensations closely related to those of cutaneous tactile sensibility, and which are caused by pungent gases and volatile substances, such as ammonia, mustard, and acetic acid. It is very probable that the sensations of smell are conveyed by the olfactory nerves, and the pungent sensations by the branches of the fifth distributed to the nasal mucous membrane.

§ 195. *Methods of Testing the Sense of Smell.*

Various substances may be employed to test the sense of smell. Care, however, should be taken not to employ substances having a pungent



odour, in order to avoid irritation of the fifth nerve. The best substances are volatile oils, as the oils of bergamont, lavender, cajeput, or cloves, and the fetid gum resins, and other substances having a penetrating odour, as camphor, turpentine, and musk.

It must be remembered that our perception of delicate flavours is due, not to the sense of taste, but of smell; hence, in testing the latter, it is necessary to employ solids and fluids which possess a delicate *aroma*, or *bouquet*, such as roast beef, cheese, or wine.

The sense of smell responds only very feebly to electric stimuli; hence the galvanic current cannot be employed as a test for it.

§ 196. *Hyperosmia*, or *Olfactory Hyperæsthesia*, occurs frequently in diseases of the central nervous system. Hysterical patients, and the subjects of various mental diseases, often complain of bad smells which are quite inappreciable to others. A bad smell sometimes constitutes the aura of an epileptic attack. These abnormal sensations depend on central irritation, and may even occur after the peripheral apparatus of smell is destroyed, so that odoriferous particles no longer give rise to the sensation.

Hysterical patients are frequently characterised by an abnormally acute sense of smell, so that they discriminate smells which are quite inappreciable to others, and distinguish various substances and even persons by the sense of smell alone.

The acuteness of the sense of smell may, like that of the other senses, be greatly increased by education. A boy, James Mitchell, who was born blind, deaf, and dumb, successfully employed the sense of smell like a domestic dog in distinguishing persons and things (M. Kendrick). Certain substances, as strychnine, either taken internally or applied locally, have the power of increasing for a time the sense of smell.

§ 197. *Hyperalgesia* of the sense of smell is frequently observed in many diseases. In this condition certain smells are regarded by abnormal feelings of like or dislike. Hysterical patients often manifest a decided aversion towards fragrant flowers, which are very agreeable to most people; while, on the other hand, they frequently show an equally unaccountable predilection for odours, like that of assafœtida, which are thoroughly disagreeable to others.

*Illusory Smells* are often perceived by the insane, although illusions of this sense are less frequent than those of the other senses. Such illusions are specially frequent in the early stage

of general paralysis of the insane. Illusions of smell sometimes occur after the use of large doses of *santonine*. *Hallucinations* of smell are also met with in the insane, these being generally of a repugnant nature. Such patients often complain of the smell of a dead body, which may lead them to think that they are surrounded by dead bodies. These conditions are probably caused by irritation of a limited portion of the cortex of the brain.

§ 198. *Anosmia, or Olfactory Anæsthesia*, consists of diminution or complete abolition of the sense of smell. When both sides are affected, the sense of taste is also considerably impaired. In *anosmia*, indeed, the impairment of the sense of taste appears more marked to the patient than in true gustatory anæsthesia, since the enjoyment derived from eating depends so much upon the perception of odorous substances and flavours. Anæsthesia of the sense of smell is a more common phenomenon in disease than hyperæsthesia. Deficiency or abolition of the sense of smell may be caused by disease of the peripheral end-organs of the olfactory nerve, of the conduction apparatus through the olfactory bulbs and brain, or of the central end-organ of smell in the cortex. The blunting of the sense of smell caused by the use of morphia is probably due to the action of the drug on the central end-organ; and the unilateral *anosmia*, which occurs in hysterical patients in association with cutaneous hemianæsthesia, is also caused either by an affection of the central end-organ or of the conduction apparatus in the brain. Abolition of the sense of smell is common in general paralysis of the insane, and it is also blunted in old age owing to senile atrophy of the olfactory nerves and of the tissues of the brain.

The sense of smell is diminished in paralysis of both the trigeminus and the facial. When the superior maxillary division of the trigeminus, which furnishes filaments to the nasal and palatine mucous membrane, is cut in animals, the mucus in the nostrils becomes thick and sanguineous; ulcerative changes may occur in the membrane, and the sense of smell is soon lost. Similar changes occur in man in disease of these nerves, producing impairment of the sense of smell. The first effect of paralysis of the trigeminus is to render the nasal mucous membrane abnormally dry, and this of itself diminishes the sense of smell by



preventing odorous particles from coming in contact with the peripheral olfactory end-organs. In facial paralysis, on the other hand, the dilators of the *ala nasi* are paralysed, so that the act of *snifing* becomes impossible, and odoriferous particles are prevented from coming into forcible contact with the mucous membrane of the olfactory chambers.

Anosmia may, indeed, be produced by anything which prevents or retards the entrance of air into the olfactory chambers; hence loss of smell is caused by constriction of the nostrils, nasal polypi, occlusion of the nasal and pharyngeal cavities. Closure of the nostrils, however, leaves the perception of flavours unimpaired, since the aromatic particles will ascend from the pharynx through the posterior nares to the olfactory chambers. Ogle relates a case in which both the sensations of smell and flavour were lost in consequence of adhesion of the soft palate to the posterior wall of the pharynx, and both sensations were restored when a communication was established, by means of an operation, between the pharynx and olfactory chambers.

§ 199. *Traumatic Anosmia*.—This form of olfactory anosmia is generally caused by blows or falls on the occiput. The elastic bones of the cranium yield to some extent to the blow; the whole of the encephalon above the tentorium is pushed forwards; the anterior margin of the temporo-sphenoidal lobe impinges against the great wing of the sphenoid bone; the olfactory nerve is apt to suffer damage at its point of junction with the brain near the anterior perforated space, or some fibres of the nerve in their passage through the cribriform plate of the ethmoid bone may be ruptured. Unilateral anosmia, caused by a blow on the occiput, is often associated with general *hemianæsthesia* of the same side, and then all the sensory disturbances are more likely to be caused by injury of the fibres of the posterior third of the internal capsule. These fibres are in the line of direction of the applied force, and are very likely to be injured by an anterior displacement of the portion of the encephalon, which is situated above the tentorium.

Anosmia of the left nasal cavity is sometimes associated with aphasia and right hemiplegia, caused by embolus of the left middle cerebral artery. Ogle asserts that in these cases the external root of the left olfactory tract is affected.

Dr. Althaus has recently described a case of anosmia, which he attributes to perineuritis of the olfactory nerves. The

affection may also be caused by tumours in the anterior fossa of the skull, or in one of the anterior cerebral lobes, by basal meningitis, or by exostoses, or caries of the bones. Anosmia is occasionally congenital, and caused by absence of the olfactory tracts (Clocquet). Senile anosmia is caused, according to Prévost, by degeneration and atrophy of the olfactory nerves.

*Prognosis.*—The prognosis depends upon the cause of the affection. Those cases which result from serious organic disease are, of course, incurable. When the loss of smell is due to coryza, exposure to cold and traumatism, the prognosis is more favourable.

*Treatment.*—The treatment of hyperæsthesia of the olfactory sense must depend upon the cause of the affection, and it is improbable that direct treatment of the olfactory nerve would be of any use. The treatment of anæsthesia must be directed against the cause of the affection; though Duchenne obtained good results from faradisation of the nasal mucous membrane, especially in hysterical patients. Beard and Rockwell also recommend faradisation and galvanisation, applied partly outside the nose and partly to the mucous membrane, by means of an electrode made in the form of a sound.

## (II.)—DISEASES OF THE SENSE OF SIGHT.

§ 200. *Structure of the Optic Nerve.*—The optic nerve commences at the chiasma and passes into the orbit through the optic foramen.

(1) *The Sheaths of the Optic Nerve* consist of (a) an external sheath of dense fibrous connective tissue, similar in structure to, and continuous with the dura mater of the brain, and named *the dural sheath* (Fig. 26, l); (b) a middle sheath, continuous with and of the same structure as the arachnoid—the *arachnoidal sheath* (Fig. 26, r); and (c) an inner sheath of fibrous connective tissue, being a continuation of the pia mater—the *pial sheath* (Fig. 26, p). Between the dural and arachnoidal sheaths is a lymph space in open communication with the subdural space of the brain, and called the *subdural space of the optic nerve* (Fig. 26, m). Between the arachnoidal and pial sheaths is another lymph space, which communicates with the subarachnoidal space of the brain, and is called the *subarachnoidal space of the optic nerve* (Fig. 26, n). The subdural and subarachnoidal spaces of the optic can be readily injected from the corresponding spaces of the brain, but do not communicate with one another.



(2) *Substance of the Optic Nerve.*—The optic nerve is composed of a large number of bundles of nerve fibres (*Fig. 26, i i*), separated from one another by trabeculae of fibrous connective tissue (*Fig. 26, k k*), containing numerous connective tissue cells, and directly continuous with the pial sheath of the optic nerve. This framework contains blood-vessels, and it forms a special accumulation around the arteria centralis. The bundles are of variable size, and are composed of medullated nerve fibres that do not possess any sheath of Schwann. The medullary sheath appears to possess at times more or less regular varicosities owing to an accumulation of fluid between the axis-cylinder and sheath (Klein).

The nerve fibres are separated from one another by a substance which is identical with the neuroglia of the spinal cord. A large lymph space may be injected on the inner surface of the pial sheath, which is continuous with lymph spaces situated within the bundles of nerve fibres and the trabeculae of the framework, and with minute lymph spaces separating the individual nerve fibres. It will thus be seen that the structure of the optic nerve is more like that of the white substance of the brain and spinal cord than that of the peripheral spinal nerves.

FIG. 26.

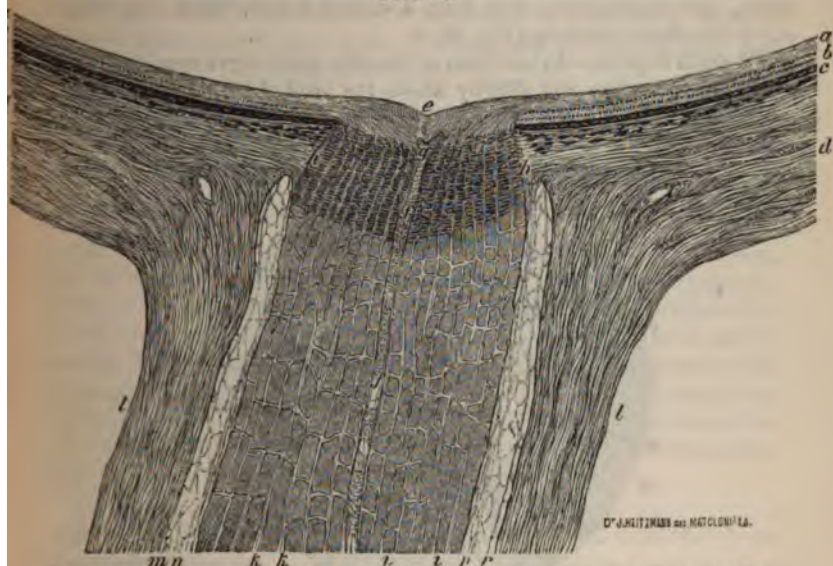


FIG. 26 (From Landolt's "Physiologie"). Horizontal Section through the Optic Nerve at its point of insertion in the globe, and its passage through the membranes of the eye.—*a*, Internal; *b*, External layers of the retina; *c*, Choroid; *d*, Sclerotic; *e*, Physiological cup; *f*, Central artery of the retina; *g*, Point of its bifurcation; *h*, Lamina cribrosa; *i*, Dural sheath; *m*, Subdural space; *n*, Subarachnoidal space; *r*, Arachnoidal sheath; *p*, Pial sheath; *i f*, Bundles of nerve fibres; *k k*, Connective tissue trabecule.

*Sclerotic Foramen and Optic Disc.*—The fibres of the optic nerve, as well as its vessels, pass through an opening in the posterior part of the sclerotic coat of the eyeball, called the *sclerotic foramen*. This opening, however, is by no means as if it were punched out, but is divided into numerous small openings by trabeculae of fibrous connective tissue derived from the sclerotic coat, the whole constituting a sieve-like layer, which has been called the *lamina cribrosa*.

*Choroidal Ring.*—The fibres of the optic nerve also pass through a round or oval opening in the choroid coat, which is called the *choroidal ring*, and the edge of which forms the boundary of the optic disc.

### § 201. *Ophthalmoscopic Appearances of the Healthy Fundus of the Eye.*

(1) *Sclerotic Ring.*—The choroidal ring is generally larger than the corresponding opening of the sclerotic, and, consequently, a narrow rim of sclerotic commonly appears within the former, which is called the *sclerotic ring* (Fig. 27, *a*).

(2) *Physiological Cup.*—The fibres of the optic nerve, immediately after passing through the lamina cribrosa, radiate in all directions to reach the retina, and consequently they form a central hollow, which has been called the *physiological cup* (Fig. 26, *e*).

(3) *Optic Papilla.*—As the fibres of the optic nerve curve over the edge of the sclerotic they rise slightly above the level of the retina, and the

FIG. 27.

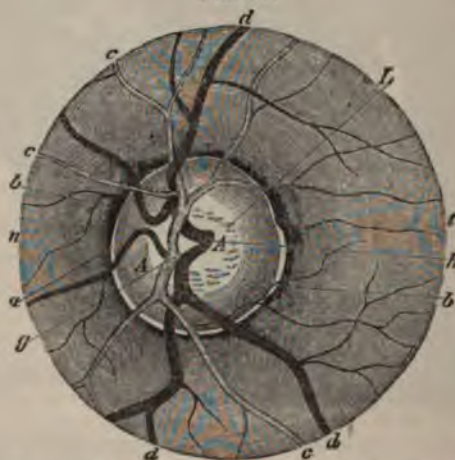


FIG. 27 (From Landois' "Physiologie," after Jaeger). *Point of Entrance of the Optic Nerve with the Retinal Vessels.*—*A A*, Optic papilla; *a*, Sclerotic ring; *b*, Choroidal ring; *c*, Arteries; *d*, Veins; *g*, Point of bifurcation of the central artery of the retina; *h*, Point of bifurcation of the central vein; *L*, Lamina cribrosa; *t*, temporal, and *n*, nasal side.



termination of the optic nerve has, from this slight prominence, been called the *optic papilla* (Fig. 27, A A).

(4) *The Retinal Vessels*.—The central artery of the retina is, as already mentioned, derived from the ophthalmic artery, while the retinal veins return the blood chiefly to the cavernous sinus; consequently, the intra-ocular circulation has been regarded as part of the cerebral circulation. The usual distribution of both artery and vein is shown in Fig. 27, g and h, and it is unnecessary to enter into a detailed description of them; although, it must be remembered that the mode of distribution of the vessels varies considerably within physiological limits in different individuals. The minute vessels of the optic disc are derived, partly from the posterior ciliary arteries, and partly from the central retinal artery, twigs from both of which commonly unite in forming the "circle of Haller," a series of vessels which surrounds the optic nerve behind the disc.

#### FUNCTIONAL AFFECTIONS OF THE SENSE OF SIGHT.

§ 202. *Optic Hyperæsthesia* consists of those conditions in which external objects are distinctly seen in an unusually dim light, or in which the acuteness of vision is very much greater than occurs in ordinary sight. The case of Sterne, who saw distinctly the satellites of Jupiter with the naked eyes, is a good example of optic hyperæsthesia. Pathological conditions, however, usually give rise to painful feelings or to complicated psychical reactions in connection with sight, and these conditions may be called *optic hyperalgesia*.

Other phenomena of vision, which may respectively be called *photopsia* and *chromatopsia*, may be placed in the category of optic hyperæsthesia. These phenomena are also observed on mechanical and electrical irritation of the optic nerves. Direct irritation of the optic nerves gives rise to sparks, flames, luminous balls, discs, rings, and to appearances like forked lightning, sometimes simply coloured, at other times containing all the colours of the rainbow. Illusions of sight often occur in cerebral affections and in various forms of insanity, and depend on central irritation.

§ 203. *Optic Anæsthesia* is characterised by diminution or abolition of vision. The anæsthesia may declare itself in the central part or by concentric diminution of the field of vision, or both conditions may be combined. Diminution of sight through an affection of the nervous apparatus is called *amblyopia*, while abolition of sight is called *amaurosis*. These conditions may be caused by lesions either of the retina, disc, optic nerves

and tracts, conduction apparatus through the corpora quadrigemina and corona radiata, or optic centres in the cortex.

The forms of amblyopia and amaurosis, which occur in the absence of any apparent lesion of the fundus of the eye on ophthalmoscopic examination, are those which interest us at present. The transitory or sometimes permanent amaurosis, which comes on after exposure to cold, venereal excess, suppression of profuse secretions, belong to this category; as well as the amblyopia which occurs after injury or other cause of irritation of the sensory branches of the trigeminus, and which may be cured by the removal of carious teeth, extirpation of tumours, or the removal of some other source of irritation. Amblyopia is also sometimes caused by irritation of remote organs, as of the abdominal organs, by worms, constipation, gastric affections, or of the uterus by pregnancy, tumours and other affections. The affection of sight in all these cases probably depends upon disturbances of the circulation caused by direct or reflex irritation of the vaso-motor nerves. An epileptic attack may be succeeded by a temporary enfeeblement of sight, which usually rapidly disappears, but may become more or less permanent when the attacks are frequently repeated. Unilateral blindness occurring in paroxysms, and generally lasting only a few minutes, is sometimes observed in the midst of perfect health, and may possibly be regarded as a vaso-motor epilepsy. I myself suffered for upwards of ten years from this affection. The blindness was preceded for a few seconds by a feeling of tension and fulness of the right eye, then the central portion of the visual field became suddenly clouded, but the darkness spread with such rapidity that the right eye appeared to have become almost instantaneously blind. This affection lasted from one to three minutes. The attack was accompanied at times by momentary and very slight confusion, but I never observed that it was followed by any symptom whatever. I could sometimes determine an attack by pressing on the globe of the eye, and I discovered accidentally that I could bring back the sight very soon by bending the head between my legs, so that the capillaries of the face got flushed; and for many years I took a considerable interest in experimenting upon what I regarded as an interesting but innocent affection. I always attributed the attack to excessive smoking; and becoming afraid ten years ago that the sight of the right eye was gradually getting weaker, I gave up smoking, and from that time till now I have not had an attack. Last year I recommenced smoking in moderation, but have had no return of the paroxysmal unilateral blindness, although I have recently had two or three attacks of what will be immediately described as scintillating scotoma. The amaurosis, which results from exhaustion, debility, hæmatemesis, profuse menorrhagia, or other severe loss of blood, is possibly caused by anæmia and subsequent serous effusion into the ventricles of the brain. The blindness in these cases is sometimes temporary, and disappears under tonic and stimulating treatment, at other times it is repeated



after every return of the bleeding, while again it may be permanent, so that the prognosis is doubtful. The ophthalmoscopic appearances are generally negative, or at most only show signs of diminished supply of arterial blood, with or without congestion of the veins. In some few cases, especially in the hæmorrhagic or scorbutic diathesis, small hæmorrhages may be observed in the neighbourhood of the macula lutea, which may be accompanied by sudden blindness. In other cases serous transudation and cloudiness of the discs are observed, followed by secondary atrophy of the optic nerves.

The amblyopia and amaurosis which follow acute diseases, and various toxic agents, are of obscure origin. The most common toxic agents which cause amblyopia are lead, alcohol, tobacco, opium, belladonna, quinine, and santonine. Some of these agents, such as lead, cause the affections of sight by inducing changes in the tissues of the brain and other parts of the nervous system, or of the vascular system.

Of the acute diseases typhus fever may be followed by amblyopia or amaurosis. The affection of sight comes on suddenly during convalescence, in the form of excentric sharply defined defect of the field of vision, sometimes in the form of central scotoma, or even as total amaurosis. The ophthalmoscopic appearances are negative, and it is probable that the affection is due to a cerebral lesion. Amaurosis is occasionally one of the sequelæ of scarlet fever, and appears to be caused by hydrocephalus interus or œdema of the base of the brain. Similar affections of vision may also result from measles, erysipelas of the head, pneumonia, and other acute diseases.

§204. *Symptoms.*—The symptoms of functional amblyopia and amaurosis are the same as those which are observed in organic diseases of the optic nerve, except that no ophthalmoscopic changes are observable in the former. Vision presents three distinct alterations in amblyopia:—1, Diminution in the acuteness of vision; 2, Alterations in the field of vision; 3, Changes in the perception of colours.

(1) *Diminution in the Acuteness of Vision.*—The patient sees objects through a mist; he has a difficulty in distinguishing minute objects, or at times may observe a dark spot in the centre of vision.

*Tests of the Acuteness of Vision.*—The acuteness of vision is usually tested by asking the patient to read print of a certain size of type, and at definite distances. The scales of test print which are usually used in England are those of Snellen, Jaeger, and Walton. In Snellen's scale the size of type is numbered according to the distance in feet, at which the print can be read by the normal eye. No. 3 of the scale can, for instance, be read by a normal eye in a moderate light at a distance of three feet. The

acuteness of vision is expressed by a fraction, of which the denominator is the number of the test type, and the numerator the distance in feet, at which it can be read. Other simple devices must be adopted when a person cannot read. The sight of each eye must always be tested separately.

(2) *Alterations in the Field of Vision.*—Restriction of the field of vision usually accompanies considerable change in the acuteness of vision. The field of vision may be altered in several ways, but the form usually observed in functional amblyopia begins at the margin of the field, and progresses concentrically until only a small central area is left.

*Scotoma.*—The first loss of vision may appear in the centre of the field, constituting a *central scotoma*. This dark spot is usually at the point of fixation; it is small at first, but gradually extends, and assumes a round, oval, or irregular form. At times, the whole of the field of vision is covered with scotomata, which appear like small points when the patient looks at a near object, but become larger when he looks at a distant object. Concentric narrowing of the field is often present. This form is observed in amblyopia, caused by the abuse of tobacco and alcohol.

*Scintillating Scotoma.*—This form of disordered vision appears at the onset to consist of a scotoma, generally affecting both eyes, though not to the same extent. The loss of sight is limited at first to a small portion of the visual field, which may be centric or eccentric; but it soon spreads, and as a rule one lateral half of the field of vision is affected. At other times the affection of vision is central, and images of surrounding objects to which the axes of vision are not directed may be visible as usual. The blindness is accompanied by spectral appearances, which as a rule become gradually developed as the black spot extends. In their simplest form they consist of a luminous border surrounding more or less completely the blind area, and widening as the latter expands (Liveing). This luminous circle or arc is subject to a rapid oscillatory movement, which has been variously described by different observers. In the more pronounced forms of the affection the luminous border assumes a zigzag outline, which has been compared to the angles of a fortification. It is also fringed by gorgeous colours, which are in continual trembling movement,



or appear to "coruscate" or to emit a "shower of sparks." The phenomenon generally lasts from a quarter to half an hour, and then gradually passes off. The few times that I have experienced this phenomenon I could only compare the luminous arc to a horse shoe rainbow with its convexity directed upwards, and without either zigzag outline or oscillatory movement. I could always see objects clearly in the lower half of the field of vision, and the partial blindness passed off in less than five minutes, without being followed by any other symptom.

The attack is, however, often accompanied by transitory impairment of cutaneous sensibility, along with tingling, numbness, or formication, deafness, loss of taste, embarrassment of speech (aphasia), momentary incoherence, transitory paresis of one of the limbs, vertigo, nausea, and it is usually followed by an attack of migraine.

Ophthalmoscopic examination of the eye during the attack has not revealed any abnormal appearances of the fundus (Liveing).

The similarity of scintillating scotoma to a visual epileptic aura, and of some of the symptoms with which it is associated, such as the transitory aphasia, to a slight epileptic attack, tend to show that the optic phenomena are caused by a discharging lesion of the cortex of the brain.

*Test of the Field of Vision.*—The most ready test of the field of vision is to direct the patient to fix one eye, the other being closed, on the corresponding eye of the operator, and the latter then moves his hand to the right, left, above and below, and at a certain distance from the fixed point as a centre. If the field of vision be limited in any particular direction, the observer will have to approach his hand nearer and nearer to the point on which the patient's eye is fixed before it comes into the field of vision, and thus any serious departure from the normal limit can be readily detected. If greater accuracy be required, the field of vision must be measured by means of the "mapping system," or by the perimeter, for a description of which the reader is referred to ophthalmological works.

(3) *Changes in the Perception of Colours.*—Particular attention has been drawn by Galezowski and others to the fact that there may be a defect in colour vision even when the acuteness of vision is very little impaired. On the other

hand, colour vision may be little affected when there is considerable limitation of the field of vision. The area of the field of vision varies for each colour. If coloured objects are moved from the centre to the periphery the first simple colour to be unperceived is green, the next red, while yellow and blue are lost near the edge of the field. If the distance at which each colour ceases to be distinguished is marked upon a chart, a series of concentric lines is obtained as shown in (*Fig. 28*).

In amblyopia the order in which the perception of the colours is lost is usually that in which the fields are arranged on the retina, the first defect being for green, then red, while blue and yellow are subsequently lost.

*Tests of Colour Vision.*—(1) It must be ascertained whether the patient can identify and name certain colours. For this purpose Galezowski's scale of colours may be employed. Eleven colours—red, red-orange, orange, orange-yellow, yellow, yellow-green, green, green-blue, blue, blue-violet, and violet—are arranged in this scale in the order in which they occur in the spectrum.

(2) According to the second method, which was originally introduced by Sir J. Herschell in the case of Dalton, the patient is asked to match a given colour from a number of others presented to him. Skeins of coloured wool are useful for this purpose.

FIG. 28.



FIG. 28 (after Gowers). Diagram showing the Fields of Colour Vision in a normal emmetropic eye on a dull day.—The fields are each rather smaller than on a bright day. The asterisk indicates the fixing point, the black dot the position of the blind spot. (Usually the blue field is larger than the yellow.)



§ 205. *Partial Optic Anæsthesia*.—There are several varieties of partial optic anæsthesia in which the ophthalmoscopic appearances may be negative. Sometimes the patient cannot see at night, a condition which is called *hemeralopia*; at other times sight is defective in daylight, and this condition is called *nyctalopia*.

*Colour Blindness*.—The most usual condition, however, is the one in which the patient suffers from colour blindness, and which, when it reaches a high degree, has been called Daltonism, after the famous chemist, who suffered from the affection. Those who suffer from colour blindness cannot see the red end of the spectrum; and the state of their vision is best explained by supposing, as originally pointed out by Sir J. Herschell in the case of Dalton, that their vision is *Dichromic*, and that the primary sensation of red is altogether wanting. The colour-blind, therefore, cannot distinguish a ripe cherry from the leaves by which it is surrounded except by its form.

#### DISEASES OF THE OPTIC COMMISSURE AND TRACTS.

The crossing of the fibres of the optic tracts has already been described. The subjoined diagram, borrowed from Charcot, explains readily the symptoms caused by lesions of these parts.

The lesions which usually affect the optic tracts and commissure are circumscribed affections of the bones and membranes of the brain or injuries of the base of the skull.

§ 206. *Symptoms*.—The characteristic feature of an affection of the optic tract is an enfeeblement or abolition of sight of one lateral half of the retina. The blind half of the retina is separated from the sensitive half by a vertical line, the limit between the two portions being sharply defined. When this condition is described with reference to the field of vision it is called *hemianopsia*, and to the retina *hemiopia*. Temporal hemianopsia, the condition in which one-half of the field of vision on the external, or temporal side of the eye, is diminished or lost, corresponds to nasal hemiopia, the condition in which the inner or nasal half of the retina is anæsthetic.

*Equilateral, or Homonymous Hemianopsia.*—When the left optic tract is compressed by a tumour (*Fig. 29, K*), the two left halves of the retina—the outer of one and inner half of the other—are cut off from the cortex, a condition which causes loss of the right halves of the fields of vision, constituting *right lateral hemianopsia*. The affected portions of the retinae are those which are associated in their functions, and the condition is called *equilateral or homonymous hemianopsia*.

*Crossed Hemianopsia.*—In crossed hemianopsia the inner or outer halves of both retinae are anæsthetic. When the lesion is situated over the centre of the commissure (*Fig. 29, T*), the inner halves of both retinae are affected. With reference to the fields of vision, this condition is called *double temporal hemianopsia*. In these cases there is a superposition of the images formed on the outer halves of the retinae, consequently the patients are often able to read the smallest print, but experience considerable difficulty in walking.

*Double Nasal Hemianopsia* is rare, and results from a double lesion (*Fig. 29, N, N*). In this condition the functional

FIG. 29.

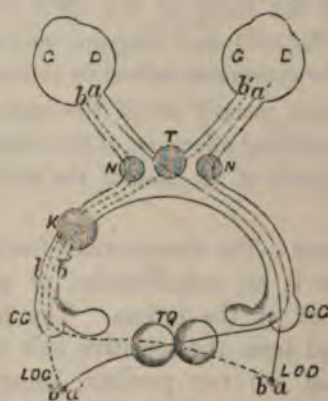


FIG. 29 (after Charcot). *Diagram of Decussation of the Optic Tracts.*—T, Semi-decussation in the chiasma; TQ, Decussation of fibres posterior to the external geniculate bodies (CG); a'a', Fibres which do not decussate in the chiasma; b'b', Fibres coming from the right eye, and coming together in the left hemisphere (LOG); K, Lesion of the left optic tract producing right lateral hemianopsia; A, lesion in the left hemisphere (LOG), produces crossed amblyopia (right eye). T, Lesion producing temporal hemianopsia; N N, Lesion producing nasal hemianopsia.



disturbances produced are great, and the patient can neither work nor guide himself. It is probable that no true hemiopia is ever caused by any intracranial disease which does not injure the commissure, or one of the optic tracts, either in its course as it winds round the cerebral peduncle, or at its origin in the external geniculate body. Conditions of vision, however, which closely simulate hemiopia, have been observed in diseases of the cerebral hemispheres, but in these cases the line which divides the blind from the sensitive portions of the retinae is never so sharply defined as in true hemiopia.

In a case under my care, and subsequently reported in "Brain," there was almost total blindness of the left, and a condition closely simulating nasal hemianopsia of the right eye. The limit between the blind and sensitive portions of the right retina was not, however, by any means sharply defined. There was a moderate degree of optic neuritis, and this symptom, along with the others present, led me to diagnose a tumour of the inferior part of the middle lobe of the cerebellum. It occurred to me that if this tumour pressed forwards on the corpora quadrigemina from left to right, it would explain the state of vision. A reference to *Fig. 29* will at once show the grounds of this conjecture. A tumour growing in the manner described would at first press on the fibres *a*, and *a'*, and then on *b*, leaving *b'* unaffected to the last. At the autopsy, six months subsequently, a glioma was found in the inferior part of the middle lobe of the cerebellum, but, instead of pressing forwards on the corpora quadrigemina, it grew along the right superior peduncle of the cerebellum, and appeared to have gradually invaded the corpora quadrigemina from right to left. Another interesting case in this connection was shown at one of the meetings of the Manchester Medical Society, by Dr. Dreschfeld. The state of vision simulated double nasal hemianopsia, but in this case also the limits between the blind and sensitive portions of the retinae were not sharply defined. This condition, along with the other symptoms, led Dr. Dreschfeld to diagnose a tumour of the inferior part of the middle lobe of the cerebellum, pressing forwards on the corpora quadrigemina exactly in the middle line, and thus interfering with the fibres *a'* and *b* in *Fig. 29*, and so causing the condition of vision present. The diagnosis has not yet been confirmed by an autopsy.

These cases, so far as they go, tend to confirm Charcot's supposition that there is a supplementary crossing of the fibres of the optic tract in the corpora quadrigemina (see *Fig. 29*, TQ). If this supposition be correct, the cortex of one hemisphere is connected with all the fibres passing from the optic nerve—not of the optic tract—of the opposite side; and destruction of the cortical centre of vision will produce, not hemiopia, but amblyopia of the opposite eye, or *crossed amblyopia*. Inasmuch as the organs of vision

are bilaterally associated in their actions, amaurosis of one eye is not produced by injury to the opposite hemisphere, although destruction of the centres of vision in both hemispheres is followed by permanent blindness of both eyes (Ferrier). Charcot has shown that in hysterical hemianæsthesia the condition of vision of the eye on the affected side is one of *amblyopia* and not of hemiopia; and a considerable number of recorded cases might be adduced to show that a similar condition is observed in hemianæsthesia from disease of the posterior part of the internal capsule, provided the external geniculate body or the optic tract of that side be not implicated. Munk, on the other hand, produced hemiopia in the monkey by removing a portion of the cortex of the occipital lobe; and recently-recorded cases appear to show that a similar condition may be produced in man as the result of disease of the occipital lobe, in the entire absence of disease of the optic tracts or external geniculate bodies. These cases have been collected and analysed by Nothnagel and by Bellouard, and both authors conclude that typical lateral hemianopsia may be caused by a lesion of the posterior extremity of the cerebral hemisphere. This subject is at present in an unsettled state, and doubtless further investigation will soon be undertaken to set the question at rest.

The prognosis of permanent hemiopia is always grave, and the treatment must depend upon the cause of the affection. Hemiopia is, indeed, not a disease of itself, but a valuable sign of other diseases.

#### DISEASES OF THE OPTIC NERVES.

The length of the optic nerves renders them peculiarly liable to suffer from secondary affections; and consequently diseases of these nerves are not only important on account of the functional disturbances produced, but on account of their diagnostic significance in cerebral and spinal affections.

##### *Diseases of the Optic Nerves (Optic Neuritis and Atrophy).*

§ 207. *Etiology.*—Optic neuritis may be caused by various intracranial diseases, and of these tumour of the brain is probably the most important.

Hydrocephalus, whether it be primary, or secondary to pressure on the veins of Galen or the lateral sinuses, also gives rise to optic neuritis. Abscess rarely occasions optic neuritis, and it is a still rarer symptom of circumscribed hæmorrhage and softening of the brain.



Basal meningitis, as well as meningeal hæmorrhages and thrombosis of the cavernous sinus, may give rise to inflammation of the optic nerves. The reason why basal meningitis occasions optic neuritis is manifest when the anatomical relation of the affected membranes to the optic tracts, commissure, and nerves is considered.

A second group of inflammatory states of the optic nerves is determined by extracranial causes. Amongst such causes must be mentioned orbital tumours, inflammatory processes in the orbit, as periostitis and cellulitis, abscesses, caries, hæmorrhage, congenital deformities, and hyperostosis producing narrowing of the optic foramina.

A third group of cases is caused by morbid conditions of the blood and consequent abnormalities of nutrition. The affections which cause optic neuritis are severe acute diseases, such as typhus, measles, pneumonia, puerperal fever, and scarlet fever. It sometimes occurs after exposure to severe cold or to a very bright light, and is occasionally produced by the sudden suppression of the menses or other accustomed discharge; or results from chronic blood poisoning by lead, alcohol, syphilis, diabetes mellitus, and, above all, from chronic Bright's disease.

#### *Varieties of Optic Neuritis and Atrophy.*

Professor Von Graefe subdivided optic neuritis into two varieties. The first is subsequent to inflammatory changes in the optic tracts or nerves within the cranium, which is propagated towards the retina; hence he called it neuritis descendens, or neuro-retinitis. In the second variety the inflammatory changes are limited to the intraocular termination of the nerve, and do not extend beyond. The optic nerve is also subject to various forms of atrophy, which may either be primary or secondary to other affections.

The diseases of the optic nerve may be divided into :—

#### *A. Congestive and Inflammatory Affections.*

1. Simple congestion of the disc.
2. Congestion with swelling of the disc (optic neuritis).

#### *B. Atrophic Affections.*

1. Simple or primary atrophy.
2. Secondary atrophy.

## (A) INFLAMMATORY AFFECTIONS OF THE OPTIC NERVE.

(1) *Simple Congestion*.—The most prominent symptom of simple congestion is increased redness of the disc. The increase in the intensity of the red colour of the disc is more readily recognised when it is greater in one eye than in the other, or greater than it was at a previous examination. The redness invades the physiological cup, and may entirely obscure it, the sclerotic ring and edge of the choroid are rendered indistinct, and the disc loses the sharpness of its outline. This is a chronic condition, and Dr. Gowers thinks that it corresponds to that described by Dr. Clifford Allbutt under the name of "chronic neuritis."

(2) *Optic Neuritis*.—In this condition œdema is associated with the congestion. The normal rosy tint of the disc is increased, its edge is blurred, and there is a pale reflection from the adjacent retina, which surrounds the disc with an indistinct halo (Gowers). The central cup is obscured, there is often distinct swelling of the disc, and it may present a striated appearance at its periphery, and the edge of the choroid is concealed. In the early stage of the affection the disc is red, swollen, and cloudy; its edge is veiled so that it cannot be seen by direct examination, and as the process increases the edge of the disc cannot be seen even on indirect examination. The disc is swollen and enlarged, its border is badly defined, and hazy, and in advanced cases its position can only be ascertained by the point of emergence of the vessels. The disc assumes a reddish grey colour and its periphery becomes distinctly striated, owing partly to the swelling and opacity of the nerve fibres, and partly to an enormous development of minute vessels. The veins are engorged, tortuous, and often varicose, while the arteries are more or less reduced in size, and appear paler than the veins. When exudation takes place, the vessels become veiled and lost to sight at the border of the disc, but reappear partially as they proceed inwards, and again disappear before reaching the lamina cribrosa. Hæmorrhages not unfrequently occur at this stage, either over the swollen disc or beyond its margin.

The inflammatory process may now subside; the swelling



gradually diminishes, the edge of the choroid becomes apparent, but some signs of the previous inflammation generally remain, such as a narrow zone of atrophy adjacent to the disc and along the edge of the choroid.

*Engorged or Choked Disc.*—If the inflammatory process proceed further, the tumour formed by the swollen papilla becomes much more prominent, and extends laterally in all directions; the margins of the swelling become steeper; and consequently, as the vessels pass over the side, they become concealed by the edge of the swelling, and reappear in a different position in the fundus (*Plate I.*). The arteries are much narrowed, and often invisible on the swelling; but the veins are usually visible towards the edge of the tumour, and often appear distended and tortuous a long distance from the disc. Hæmorrhages are now frequent and extensive, and generally appear at the edge rather than on the surface of the swelling. Sight usually disappears rapidly in the stage of strangulation. This form of optic neuritis constitutes the *choked or engorged disc* (Allbutt) *stauungs-papilla*.

*Subsidence of Neuritis.*—After the strangulation has existed for some time the venous distention gradually lessens, the swelling loses its intense red colour, hæmorrhages cease, and the extravasated blood disappears; the tumour lessens in height and extent. The most prominent part of the swelling becomes pale, and this central pallor extends laterally and gradually invades the sloping sides of the tumour and the adjacent retina. The centre of the swelling soon presents a distinct depression, and the vessels become more and more constricted owing to the cicatricial contraction of the newly-formed connective tissue. The edge of the choroid and sclerotic now become dimly apparent, and the disc has a white appearance, but does not for a long time present any central depression, although this appearance may be ultimately produced. Although the disc is white at first, it may, on reaching the retinal level, assume a faint grey tint, and as the contraction increases this tint becomes more and more marked. The edge of the disc is often irregular, and surrounded by a zone of choroidal atrophy. Retinal hæmorrhages are usually absorbed during this regressive process; but these sometimes become

transformed into spots of pigment, or lead to the formation of white spots on the retina.

*Retro-bulbar Neuritis.*—This variety of neuritis is, according to Dr. Gowers, a mixed condition of congestion and atrophy, the primary stage of congestion soon passing on to atrophy with narrowed vessels.

*Retro-bulbar Perineuritis.*—This variety is the result of chronic inflammation of the sheath of the nerve, causing thickening and purulent infiltration among the trabeculæ, and generally ending in optic neuritis. This form has been observed in periostitis of the orbit, and in constriction of the optic nerves from thickening of the cranial bones.

*General Symptoms.*—In many advanced cases of optic neuritis, even in the choked disc, sight is quite unimpaired, and when amblyopia is present its degree is by no means proportional to the amount of changes observed on ophthalmoscopic examination.

In *retro-bulbar neuritis*, on the other hand, a high degree of unilateral or bilateral amblyopia may precede for some time any ophthalmoscopic appearances.

In all severe cases of optic neuritis, diminution of the acuteness of vision, which may take place in any of the zones of the visual field, appears after a longer or shorter time. The point of distinct vision is at times excentric, while at other times there is general or partial and usually irregular contraction, or blind spots (scotomata) may occur in the visual field. At times there is great sensitiveness to light, or the patient complains of subjective sensations of sight, such as sparks or flashes of light, and coloured spectra. These phenomena may occur when there is complete blindness. The patient may also suffer from *muscæ volitantes*, and slight pain in the eyeball, caused either by irritation of the fifth or pressure on the optic nerve.

The cerebral symptoms most likely to be associated with optic neuritis are headache, vertigo, vomiting, loss of memory, epilepsy, hemiplegia, and paralysis of some or all of the ocular muscles.

*Morbid Anatomy.*—In order to understand the mechanism by which the swelling of the disc is caused in optic neuritis,



the anatomical relations of the nerve must be kept in mind (§ 199). Inflammation of the optic nerve is accompanied by a serous infiltration which augments its volume and diminishes its consistence. The sheath of the nerve is often distended with fluid, the distention being greatest a short distance behind the eye. The connective tissue of the pial sheath and of the trabeculæ surrounding the nerve bundles becomes thickened, and manifests an increase of nuclei or cells, and a considerable number of leucocytes may be observed around the vessels. A like increase of nuclei may be observed between the nerve fibres themselves. The nerve fibres become irregularly thickened, and present a varicose appearance, while the medullary sheath becomes granular, and the myeline is often broken up into globular masses. In advanced cases, the tissue of the *lamina cribrosa* becomes distended, and its structure greatly altered. The veins are large and tortuous, while the arteries are abnormally small and often quite atrophied.

The first explanation of the conditions observed in optic neuritis was attempted by Von Graefe. The condition which he designated as descending neuritis was, in his opinion, caused by inflammation communicated to the optic nerve from inflamed meninges. That the optic nerve is liable both to perineuritis and to descending neuritis, like other nerves, is not seriously doubted by any pathologist; the only question which arises is, whether the cases described by Von Graefe as descending neuritis can be clinically and anatomically distinguished from forms of optic neuritis, which arise as the result of a different mechanism. The cases of optic neuritis or choked disc, which occur in association with cerebral tumour, and in which there is no evidence of inflammation in the trunks of the optic nerves, Von Graefe attributed to increase of intracranial pressure, obstructing the return of blood from the eye by compressing the cavernous sinus, an obstruction which would be greatly intensified by the unyielding character of the sclerotic ring. It was, however, demonstrated by Sesemann that the supra-orbital anastomoses so freely with the facial veins that pressure on the cavernous sinus would not produce anything beyond a temporary effect. It was discovered by

Schwalbe that the subvaginal space around the optic nerve is continuous with the subdural space around the brain; and Schmidt suggested that any increase of intracranial pressure would tend to distend the sheath of the optic nerve with fluid, and consequently would produce strangulation of the nerve fibres in their passage through the sclerotic ring and lamina cribrosa. Although this theory is very largely accepted, it is by no means free from objections. In the first place, optic neuritis is rarely found in chronic hydrocephalus, where the intracranial pressure is very great; while the affection may be present in an intense degree in the case of a small tumour, where the increase in the intracranial pressure must be relatively slight. Whatever part, therefore, increase in the intracranial pressure may take in the production of optic neuritis, it is clear that some other factor is necessary for the full explanation of the mechanism of its production. This factor Dr. Hughlings Jackson endeavoured to supply by suggesting that intracranial tumours act like foreign bodies, and produce optic neuritis by their irritating effects; but, as Dr. Clifford Allbutt remarks, the evidences of irritation are frequently absent in the tissues surrounding intracranial tumours, although such cases during life were accompanied by optic neuritis.

*Treatment.*—Idiopathic optic neuritis must be treated by active antiphlogistic measures. Two or three leeches should be applied to each temple, or a few ounces of blood may be removed from the temple by the artificial leech. In the case of optic neuritis from cerebral tumour the possibility of syphilis must never be forgotten, and it is a good rule to administer the iodide of potassium in all cases in which there is the slightest possibility that this disease may have been acquired. The iodide should be given in large doses to begin with, and the dose still further increased if there is the slightest sign of improvement.

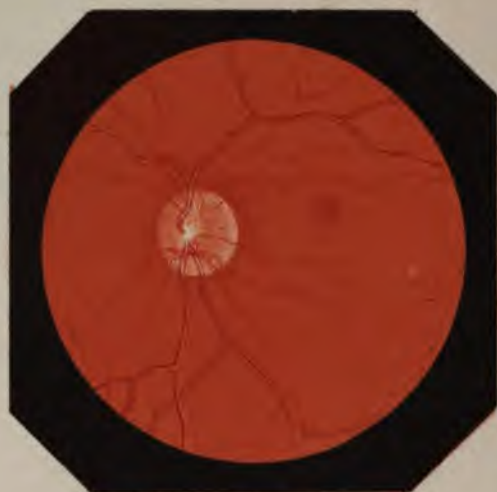
#### (B) ATROPHIC AFFECTIONS OF THE OPTIC NERVES.

*Atrophy of the Optic Nerve.*—Atrophy of the optic nerve is a state in which the intraocular extremity of the nerve becomes slowly and progressively transformed into a pure white or greyish-white disc. There is complete cessation of the capil-





Plate 1.



Normal fundus of Eye.



Optic Neuritis.



White disc Atrophy.

*E. Burgess del.*



Grey disc Atrophy.

*West, Newman & Co. Lith.*



lary nutrition of the nerve, and consequently the healthy rosy tint of the disc disappears.

(1) *Simple atrophy* is frequently associated with disease of the spinal cord, and especially with locomotor ataxy; and consequently Charcot called it *tabetic amaurosis*, or *parenchymatous atrophy*. It appears at various epochs in the course of the disease. Sometimes it is preceded by the lancinating pains in the inferior extremities, and by many of the other symptoms of the disease; while, at other times, the optic lesion precedes by many years all the other symptoms. As this affection comes on without any definite cause, it may also be called *primary atrophy*.

(2) *Secondary atrophy* of the optic nerve is caused in various ways. The following varieties of the affection may be distinguished:—

(a) *Atrophy by compression of the fibres of the Optic Nerve*. This form of atrophy may be caused by pressure on the chiasma or on the optic nerves in any part of their course through the base of the brain, optic foramen, or orbit. It is often preceded by a stage in which the disc is congested; but inasmuch as the ophthalmoscopic appearances presented by the simple and congestive varieties are the same when once atrophy is established, the two forms may be described together. Lesions of one optic tract cause bilateral symmetrical hemiopia, but it is only after some years that slight pallor of the corresponding halves of the disc is observed.

*Symptoms: Ophthalmoscopic Appearances*.—On ophthalmoscopic examination the optic disc is observed to have undergone very marked changes. (See *Plate I*.) The disc assumes a pearly white colour (white atrophy), occasionally mixed with a slight tinge of blue or of grey (grey atrophy). The contour of the disc is sharply defined; but its outline may be slightly irregular, and in advanced cases it often becomes oval and somewhat reduced in size, although the normal size and round form may be long preserved after the fullest blanching. The central artery and vein maintain their normal volume and direction, but the lateral branches of the disc are in great part atrophied. This affection is usually bilateral, but occasionally it may be limited for many years to one eye, and become

only slowly communicated to the other. The retina remains perfectly transparent, and preserves its normal aspect, the disc being the only part of the fundus which presents any appreciable change. The pupil is usually contracted (*myosis*), but at other times it assumes an irregular, angular or oval form, probably caused by atrophy of some of the branches of the ciliary nerves. The pupils are also often of unequal size.

*General Symptoms.*—The onset of the affection is usually slow, and the patient observes for months or years that his sight is becoming gradually and progressively enfeebled. The field of vision is not as a rule diminished at first, but with the progress of the affection it becomes concentrically contracted. Defect in the acuteness of vision is usually, but not always, in direct proportion to the degree of change in the optic nerve, and occasionally the patient can read the finest print when the atrophy is very advanced, and when there is concentric diminution of the field of vision. The latter may be altered in various ways, but concentric limitation is the most frequent change. At times a central scotoma may be associated with concentric restriction. This form of alteration generally arises from toxic causes, such as alcohol and tobacco. Loss of colour vision (*achromatopsia*) is especially well marked in this form of atrophy.

Various subjective sensations are experienced, such as sparks or flashes of light (*photopsia*), or a subjective play of colours or coloured spectra (*chromatopsia*). The mode of locomotion of these patients is characteristic. The head is retracted and the chin elevated, the gait is shuffling, the eyes are directed upwards, and the expression of the countenance is vague, owing to the fact that the eyes are not fixed on any object. If the affection has been developed slowly, nystagmus may be present.

(b) *Atrophy Secondary to Optic Neuritis.*—The main features of this form of atrophy have already been described, when the subsidence of neuritis was under consideration. The disc is at first yellow or dull white, its contour is completely hidden under an exudation, the vessels are varicose; but as the exudation becomes absorbed the nerve becomes whiter and whiter, its capillaries atrophy, and the central vessels themselves become smaller although they preserve their tortuous



course. The disc is not sharply defined, as in simple atrophy; but its contour is irregular and broken, and its edges are obscured by exudation. It is larger than normal, and white patches of exudation may be observed in its vicinity and in the neighbourhood of the macula, indicating that the inflammation had previously spread to the retina.

Atrophy secondary to optic neuritis is not always followed by blindness, and patients who had completely lost their sight during the acute period may now recover some degree of vision and retain it for the remainder of life.

(c) *Atrophy Secondary to Obliteration of Vessels.*—This form of atrophy is caused by embolus or thrombosis of the central artery of the retina. The disc is of a pearly white colour without a tinge of grey; but its margin is usually covered with a white veil, which extends to the retina. The arteries are so small as to be scarcely appreciable, and are often surrounded by a whitish and more or less opaque exudation. After the exudation has been absorbed, this form of atrophy is distinguished from every other kind by the small size of the arteries of the disc and retina.

(d) *Choroiditic Atrophy.*—Atrophy of the disc secondary to choroido-retinitis closely resembles that which results from obliteration of the central artery of the retina. There is a marked wasting of the retinal vessels in choroiditic atrophy; but the disc is often characterised by a peculiar reddish or yellowish tint, and its edges appear slightly blurred (Gowers).

(e) *Atrophy Secondary to Retinitis Pigmentosa.*—In retinitis pigmentosa the retinae become studded by pigmentary spots. This migration of the choroidal pigment into the retinae is accompanied by an atrophy of the central vessels, their walls becoming thickened, and their calibre much diminished in size. On ophthalmoscopic examination the central vessels appear sensibly diminished in size, and their collateral branches undergo the same alterations, and after a time disappear altogether. The capillary vessels derived from the ciliary arteries of the optic nerve are, however, not affected to the same extent; hence the disc generally preserves a well marked rosy tint.

(f) *Atrophy by Excavation.*—This variety of atrophy is

caused by increased intraocular pressure in such diseases as glaucoma and hydrophthalmia, and it is characterised by the deep excavation of the optic disc. A certain amount of excavation may take place in the later stages of any form of atrophy, owing to wasting of the fibres of the optic nerve, and to cicatricial contraction of newly-formed connective tissue.

§ 208. *Morbid Anatomy.*—The anatomical changes in atrophy of the disc extends through the optic nerves. In primary atrophy the nerve is much reduced in size, and is grey and gelatinous in appearance. The connective tissue trabeculae are hypertrophied, while the nerve fibres themselves are progressively destroyed. During the disappearance of the medullary sheath, which is the first to undergo degeneration, the various transformations of myeline already described in the case of other nerves may be observed. After a time the axis-cylinder also becomes destroyed, and the nerve becomes changed into a cord of connective tissue. In atrophy from pressure the nerve is much reduced in size, and there is great increase of connective tissue. The disc usually presents a superficial depression, which, according to Müller, does not generally pass beyond the limits of the choroid. The lamina cribrosa does not generally undergo any displacement, and it is only covered by a thin layer of the debris of the disc. Müller has also shown that the ganglionic layer and the nerve fibres of the retina undergo complete atrophy, while the other layers are unaffected. The degeneration always ascends to the chiasma, and the optic tracts are atrophied in long-standing cases as far as the external geniculate bodies.

§ 209. *Course and Terminations.*—The progress of the affection is usually slow, and from three to six years may elapse before complete blindness comes on. Syphilitic atrophy of the optic nerve is more rapid in its progress, and it may cause blindness in the course of a few months. The atrophy is limited at times to one eye, but as a rule it is binocular.

§ 210. *Prognosis.*—Whatever may be the form of atrophy of the disc, it is undoubtedly one of the gravest affections which



can affect the organ of vision. In progressive atrophy recovery is almost entirely unknown. A temporary improvement may at times be obtained, but it is doubtful how far this is due to treatment, inasmuch as the functional symptoms may remit slightly, even where the atrophic process is steadily progressive. The prognosis of atrophy secondary to optic neuritis is more favourable. The cerebral affection which has caused this affection may be capable of cure, and the atrophy may become partial and cease to progress further.

Monocular atrophy of the disc, provoked by a local cause, ocular or orbital, is less grave; the lesion being local, the atrophy remains limited to a single eye, and the sight of the other is preserved.

§ 211. *Treatment.*—The treatment of atrophy of the optic nerve consists in removing the exciting causes of the affection, and the general condition of which it is a symptom. It is necessary to inquire whether there may be some debilitating cause for the disease, such as exhausting diarrhœa, excessive menstrual or hæmorrhoidal discharge, and sexual or any other form of mental or physical excess calculated to engender nervous debility. An inquiry must also be instituted to ascertain whether the disease depends upon the abuse of alcohol, tobacco, or other toxic agents.

The greatest care must be taken to ascertain if the disease is due to a syphilitic taint, either acting primarily on the nerve or its vessels, or secondarily through the optic neuritis caused by the growth of a gumma within the cranium. The slightest suspicion of such a cause must be promptly followed by appropriate antisyphilitic treatment. Atrophy from scrofulous disease of the brain must be treated by iodide of iron and cod-liver oil, although the prognosis in this case is not so favourable as in the syphilitic variety. Nitrate of silver, phosphorus, and strychnia have each been found useful in the treatment of this affection, the first of them being probably most appropriate for the treatment of the tabetic variety. Electrification of the eye is occasionally of use in the treatment of cases in which vision is not completely lost.

## (III.)—DISEASES OF THE ACOUSTIC NERVE.

§ 212. *Auditory or Acoustic Hyperæsthesia* is that condition in which the sensibility of the auditory sensory apparatus is abnormally increased; so that sounds too far off to be detected in the normal state are distinctly heard, or the power of discriminating differences of sounds is greatly increased. Great differences obtain in health in the degree of acuteness of the sense of hearing in different individuals or in the same individual at different times. Like the other senses, the ear is capable of being educated; and trained musicians can detect the most delicate shades of differences in tone, and it is a matter of common observation that the acuteness of hearing is greatly increased in the blind. The increased power of distinguishing various sounds Eulenburg proposes to call *hyperakusia*. *Hyperakusia* is often observed in hysterical patients and in conditions of ecstasy and sometimes even in somnambulism. *Hyperæsthesia* of the sense of hearing, however, generally declares itself, not by an increased power of discriminating sounds, but in an increase of the pleasant or painful feelings which accompany sound. This condition may be called *auditory hyperalgesia*. The painful feelings associated with sound predominate in disease. *Auditory hyperalgesia* is a very troublesome symptom in many cases of acute disease, general debility, hysteria, and various mental diseases, so that the slightest sound is exceedingly painful to the patient.

*Auditory hyperæsthesia* may be caused by an affection of the peripheral endings of the auditory nerve, of the conductive apparatus, or of the central acoustic centre, and it may also occur as a symptom of disease of the accessory apparatus of hearing.

*Auditory hyperæsthesia* occurs in peripheral facial paralysis, due to increased tension of the tympanic membrane from paralysis of the stapedius muscle.

The condition in which the sensation of hearing is accompanied by painful feelings must be carefully distinguished from *auditory neuralgia* or *otalgia*. *Otalgia* in all probability depends upon neuralgia of filaments of the inferior maxillary branch of the trigeminus, and it has been called by Kramer and Schwartz neuralgia of the tympanic plexus.



§ 213. *Concomitant Symptoms*.—Acoustic hyperæsthesia is often accompanied by auditory subjective sensations, illusions, and hallucinations. *Tinnitus* is the most common form of subjective sensation, and it may assume the forms of clanging, humming, whizzing, whistling, and other noises. This condition is seldom of nervous origin. It is generally a symptom of the most different diseases of the external or middle ear. Tinnitus is sometimes a symptom of congestion of the head, and occurs in association with dizziness in Meniere's disease, also in anæmia and chlorosis, after great loss of blood, and after the use of large doses of quinine, or salicine.

The constant current is a very valuable agent in diagnosing different kinds of tinnitus, and also very successful in the treatment of the neuropathic forms. In neuropathic kinds there is hyperæsthesia to the galvanic current during cathodal closure, anodal opening, and the closed circuit in both ears. In some cases an anomalous reaction is obtained in the opposite ear to that to which the electrode is applied. In those moments of changing the current which produce no effect on the ear, irritative sensations are heard in the other ear similar to those which would be heard if it were directly irritated by the opposite direction of current.

Auditory illusions are frequently observed in mental diseases, probably not so often as illusions of sight, but frequently in association with the latter.

The illusions depend on irritation of the central acoustic organ. Auditory illusions are most frequently observed in melancholia and craziness. In the former the illusions assume the form of abusive or threatening words, or commands to do acts of violence; in the latter they take the form of heavenly messages, revelations, or the solace of the presence of sweet music. Such phenomena are not unfrequently associated with complete deafness. Auditory illusions are sometimes unilateral; and in some cases there are alternating illusions of different senses, such as right-sided optic and left-sided auditory illusions.

§ 214. *Auditory Anæsthesia* consists of abnormal diminution or abolition of the sense of hearing. The cause of the affection may be either lesion of the peripheral end-organ of the auditory apparatus, of the conductive apparatus, or of the central terminal organ. Dulness of hearing or complete deafness may occur after various injuries to the head, such as fracture of the base of the skull, severe concussions, or a fall on the back of the

head. Deafness is a symptom of circumscribed affections of the cerebellum, and of the middle and posterior lobes of the cerebrum; also of cerebro-spinal and basal meningitis, and it is frequently a symptom of acute infectious diseases, such as scarlet fever, measles, and typhus. Deafness is observed in hysteria, and after toxic doses of quinine, lead, and other agents. The consideration of the pathology of these conditions must be deferred for the present.

Deaf mutism is usually a congenital affection, and it generally depends on arrest of development of the internal or middle ear; sometimes it is acquired.

*Treatment.*—The treatment of acoustic hyperæsthesia and anæsthesia has not been attended by great success. Acoustic hyperæsthesia is sometimes improved by galvanisation, moderately strong currents being employed. Duchenne obtained good results in nervous deafness from the faradic current.

#### (IV.)—DISEASES OF THE GUSTATORY NERVES.

§ 215. The tongue is the principal organ of the gustatory sense; but sensations of taste are also perceived by part of the soft palate, by the arches of the palate, and by the walls of the pharynx. But the root of the tongue and the pharyngeal tissues on the one hand, and the anterior half of the tongue on the other, receive their gustatory fibres from different nerves. It may be accepted as proved that the glosso-pharyngeal nerve supplies the gustatory fibres distributed over the posterior part of the tongue, the palate, and the walls of the pharynx, and by the mutual contact and pressure of these parts the sensations are rendered more acute and distinct. The tip and anterior two-thirds of the tongue receive their gustatory fibres from the lingualis. It has been proved that section of the chorda tympani, or of the facial nerve at any point where it contains the fibres of the chorda tympani, also abolishes or retards the perception of taste in the anterior part of the tongue. Whether all the gustatory fibres pass from the lingualis to the chorda tympani, or part of them remain in the lingualis, is a question which has not yet been finally settled; but there can be no



doubt that the chorda tympani contains a very considerable part, if not all, of the gustatory fibres of the anterior half of the tongue.

FIG. 30.



FIG. 30 (From Hermann's "Physiology"). *Diagram of Glosso-pharyngeal Nerve, its connections and branches.*

GP, Glosso-pharyngeal nerve. JG, Its jugular ganglion. PG, Its petrous ganglion.

1, Tympanic branch, or nerve of Jacobson, the branches of which are as follows:—  
2, Filaments to plexus on carotid artery; 3, To Eustachian tube; 4, To fenestra rotunda; 5, To fenestra ovalis.

6, Twig of union with small superficial petrosal n.

7, Twig of union with great superficial petrosal n.

8, Pharyngeal branches of glosso-pharyngeal n.

9, Muscular branches to stylo-pharyngeus and constrictors (?)

10, Tonsillitic branches.

11, Terminal lingual branches.

P, Pneumogastric nerve, from the ganglion of the root of which branches pass to the petrous ganglion.

S, Superior cervical ganglion with an ascending branch to the petrous ganglion.

MG, Meckel's ganglion. OG, Otic ganglion. F, Facial nerve. CA, Carotid artery.

FIG. 31.

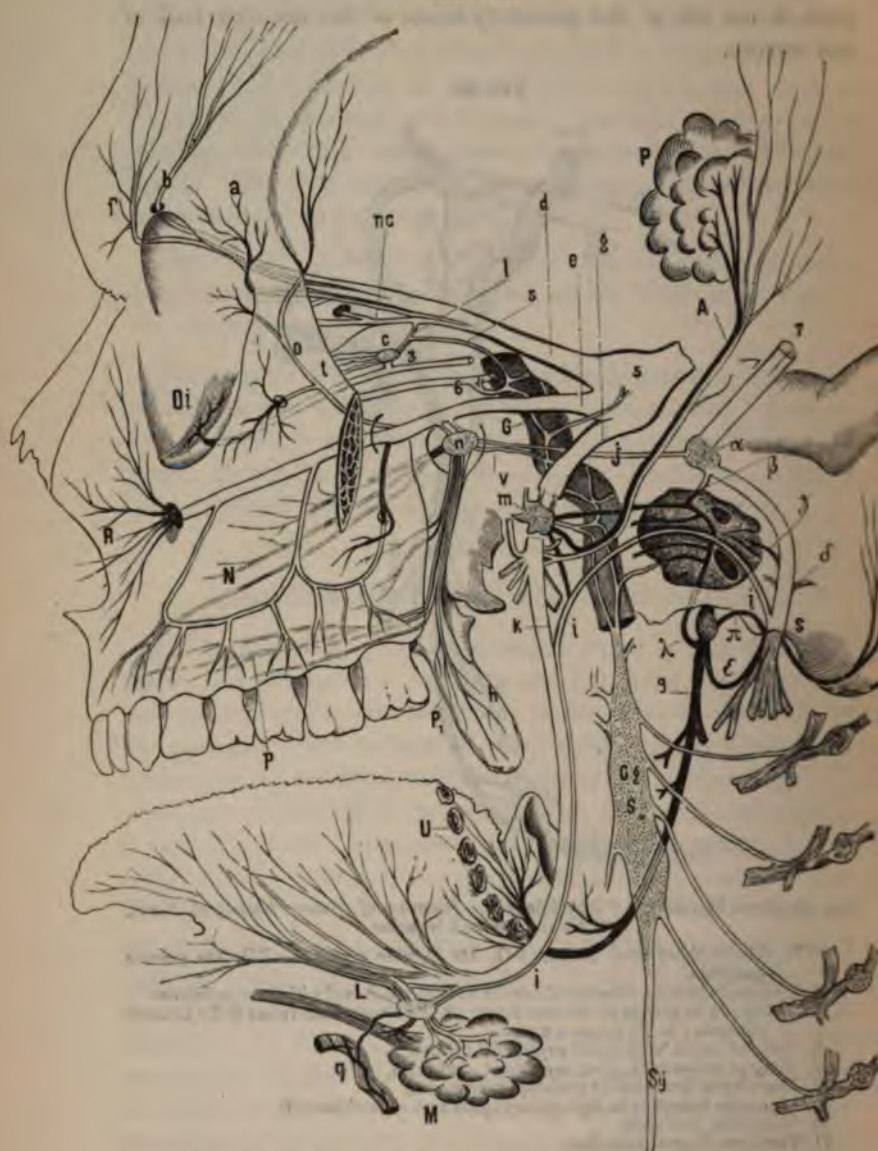


FIG. 31 (From Landois' "Physiologie"). Semi-schematic representation of the Ocular Nerves, along with the Trigeminal and its Ganglion, the Facial, and Glosso-pharyngeal Nerves.—3, Branch of the oculo-motorius to the inferior oblique muscle, with a thick short root to the lenticular ganglion (c); t, ciliary nerves; t, long root of



Schiff found that section of the trigeminus at the base of the skull completely abolishes the sense of taste in the anterior half of the tongue, and the same result follows the division of the great superficial petrosal nerve, the division of all the connections of the spheno-palatine ganglion, and the extirpation of the spheno-palatine ganglion itself. The clinical records relied upon in determining the respective functions of the nerves distributed to the tongue and mucous membrane of the pharynx consist of—

1. Cases of complete isolated anæsthesia of the trigeminus from disease of the segment lying at the base of the skull, with abolition of taste in the anterior half of the tongue.

2. Cases of isolated complete peripheric paralysis of the facial, with abolition of taste on the anterior half of the tongue. Also cases of suppuration of the middle ear or caries of the temporal bone, with paralysis and lesion of the chorda tympani.

3. Cases of paralysis of the facial at the base of the skull, above the geniculate ganglion, without injury to the sense of taste (Erb).

A considerable number of such cases are now recorded, and it may consequently be inferred, that the greater part of the gustatory fibres pass from the lingualis to the chorda tympani, and by means of the latter into the facial, but finally return again into the trigeminus, with which they enter the brain.

So far, then, as our present knowledge enables us to judge, the following is the distribution of the gustatory nerves. The base of the tongue, the palate, and the walls of the pharynx are supplied by the glosso-pharyngeal nerve; the anterior two-thirds of the tongue, on the other hand, is supplied by the lingual, but the gustatory fibres of that nerve, either altogether or in great part, enter the chorda tympani, then run in the facial as far as the geniculate ganglion, and finally return by many and still imperfectly known paths to the second and third divisions of the fifth nerve, in which they ascend to the brain.

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the ganglion from the nasal nerve (*nc*); *s*, sympathetic root from the internal carotid plexus (*G*); *d*, first branch of the trigeminus (*5*) with the nasal (*nc*) branch and the terminal branches of the lachrymal (*a*), supra-orbital (*b*), and frontal (*f*) nerves; *e*, second branch of the trigeminus; *R*, supra-orbital; *n*, spheno-palatine ganglion, with its roots (*j*) from the facial, and (*v*) from the sympathetic; *N*, the nasal branch; *p*, *p*, the branches of the ganglion to the gums; *g*, third branch of the trigeminus; *k*, lingualis; *i*, *i*, chorda tympani; *m*, otic ganglion, with its roots from the tympanic and carotid plexuses, and from the third branch of the fifth; and with its branches to the auriculo temporalis (*A*) and to the chorda tympani (*i*, *i*); *L*, submaxillary ganglion, with its roots from the chorda tympani, lingualis, and the sympathetic plexus of the external maxillary artery (*g*); *7*, facial nerves; *j*, superficial petrosal (major) nerve; *a*, geniculate ganglion; *β*, branch to the tympanic plexus; *γ*, branch to stapedius; *δ*, communicating branch to auricular branch of the vagus; *i*, *i*, chorda tympani; *s*, stylo-mastoid foramen; *9*, Glosso-pharyngeal nerve, *λ*, its tympanic branch, *π* and *κ* communicating branches to facial nerve, *U*, termination of the gustatory fibres in the papillæ circumvallatæ; *Sy*, sympathetic with (*Gy Sy*) the superior cervical ganglion; *i*, *ii*, *iii*, *iv*, the four upper cervical nerves; *P*, parotid; *M*, submaxillary gland.

§ 216. *Methods of Testing the Taste.*

It ought to be remembered that many things differ considerably in taste according as they are perceived by the anterior or posterior portion of the tongue, and that no substance can be tasted unless it is dissolved.

When the acuteness of the sense of taste is to be tested, the patient should be directed to put his tongue out with the mouth widely open and the eyes closed, whilst the sapid substance is applied on the particular part to be tested with a glass rod or small brush. The tongue must not be withdrawn into the mouth until time is given for the taste to be perceived, the greatest difficulty being experienced in keeping the stimulus circumscribed to a particular part of the tongue. After each experiment the tongue must be prepared for a fresh trial by rinsing the mouth out with water.

A solution of quinine, infusion of quassia, or solutions of various other substances are employed for testing *bitter tastes*; and it ought to be remembered that bitters are most distinctly perceived at the root of the tongue. Solution of sugar, honey, and so on, are used for testing the taste for *sweet* articles, which are most distinctly perceived at the tip of the tongue. Vinegar and diluted acids are used for testing *acid tastes*, which are chiefly perceived by the edges of the tongue. *Saline tastes* are tested with solutions of common salt, bicarbonate of soda, and the like.

The galvanic method of testing the sense of taste, introduced by Neumann, is very valuable for the investigation of pathological cases. Two fine wires, provided with small knobbed ends, and carefully isolated from one another by means of sealing-wax, are to be attached at a few millimetres from each other to a non-conducting handle, such as a glass rod or an elastic catheter, and these wires, which form the electrodes, are then connected with the poles of one or several galvanic elements. If these be placed upon the dorsum of the tongue, a slight burning sensation is felt with a distinct sensation of taste, which is stronger at the anode than at the cathode, and which is variously described as being sourish, saline, metallic, &c. The limits of the gustatory and non-gustatory areas may be accurately determined by this means.

The neuroses of the gustatory nerve may be subdivided into two groups, the first of which includes the hyperæsthesiæ and the paræsthesiæ, and the second the various forms of anæsthesia.

§ 217. *Hyperæsthesia of the Gustatory Nerves.*—Hyperæsthesia of the sense of taste may manifest itself as an increase in the delicacy of the taste. Hysterical patients sometimes detect certain ingredients in food or medicines which are quite imperceptible to healthy persons. It may again express itself as an increase in the enjoyment or loathing of food, certain sub-



stances causing a more agreeable or disgusting taste than they do in the case of the healthy palate. These anomalies chiefly occur in hysterical patients. Amongst the *paræsthesiæ* of the sense of taste may be mentioned the subjective sensations which are perceived in the anterior half of the tongue in many cases of facial paralysis of rheumatic origin, and which are described as being sourish, sweetish, or insipid in taste; and the gustatory sensations which are perceived in the tongue when certain drugs have been taken. The bitter taste after the use of *santonine*, for instance, is so strongly marked that water seems to have a bitterish taste when swallowed, and an intensely bitter taste is often experienced during attacks of jaundice. The subjective gustatory sensations of insane patients are, doubtless, of centric origin, and consist partly of hallucinations, and partly of illusions of taste.

§ 218. *Anæsthesia of the Gustatory Nerves* may be, as regards its intensity, either complete or incomplete, and the diminution or loss of taste may include all the varieties of sapid qualities, or only a few of them. *Jacobowitch* has described the case of a patient who was unable to perceive either bitter or acid flavours, but, on the other hand, recognised sweets and salines perfectly. The anæsthesia of the gustatory sense may also be circumscribed or diffused, affecting either the tip of the tongue, its root, or one or both sides.

Peripheral gustatory anæsthesia may be caused by all conditions which prevent or render difficult the action of sapid substances upon the terminal organs of the gustatory nerves. The most usual of these conditions are a coating of thick fur, and preternatural dryness of the mucous membrane. Agents which lower the excitability of the terminal organs also blunt the sense of taste, such as cold and even excessive heat, for it is well known that the taste of hot foods is not recognised with precision.

The most usual cause of gustatory anæsthesia is to be found in disease of the conducting fibres. The lesion may be situated in the course of the glosso-pharyngeal, trigeminal, lingual, chorda tympani, and facial nerves. When the glosso-pharyngeal is affected, taste is weakened or abolished on the corresponding

side of the root of the tongue, palate, and pharynx. No uncomplicated case of this nature is however on record. If the lesion is situated in the *trigeminus* and *lingualis*, or in the *chorda tympani* and certain sections of the facial, the gustatory anæsthesia affects the anterior half of the tongue, its borders and apex. Many cases are on record where intracranial lesion of the trigeminus caused paralysis of the sense of taste; and Romberg relates a case where the third branch of the trigeminus was alone diseased, and where there was gustatory anæsthesia.

§ 219. *Lesions of the chorda tympani*, caused by disease of the internal ear and caries of the temporal bone, have been frequently observed to produce paralysis of the sense of taste in the anterior half of the tongue. Lesions of the facial nerve have also been found associated with gustatory anæsthesia in the anterior half of the tongue on the same side. The lesion is probably situated within the Fallopian canal and below the geniculate ganglion. Cases are recorded where division of the facial immediately below the stylo-mastoid foramen was followed by gustatory impairment; but it is probable that the chorda tympani might also have been injured in some of these cases. The influence of intracranial lesion of the facial nerve on the sense of taste has not been thoroughly investigated, but Erb met with a case where the concomitant symptoms pointed to intracranial lesion of the nerve, and where taste was unaffected. Our knowledge of central anæsthesia of the gustatory sense is very imperfect. If the areas of distribution of the lingual and glosso-pharyngeal nerves are coincidently rendered anæsthetic, and if there be also extensive muscular and cutaneous anæsthesia, it may be inferred that the cause is a central lesion. Such cases are generally met with in hysteria, and then loss of taste is frequently associated with hemi-anæsthesia. Nothing is known of the effect of disease of the perceptive centres on taste.

*The diagnosis* must be made by a careful objective investigation, according to the methods previously described.

*The prognosis* depends upon the nature of the primary disease.



*The treatment* must be mainly directed to remove the cause of the disease, but direct treatment may be associated with casual treatment. The best direct treatment is the application of faradisation or galvanisation, either directly to the tongue itself, or through the lingual nerve, or the current may be passed through the head, the poles being applied over the temples or the mastoid processes.

## CHAPTER IV.

## DISEASES OF THE MOTOR CRANIAL NERVES—(OCULAR, FACIAL, AND HYPOGLOSSAL NERVES).

## (I).—DISEASES OF THE OCULAR NERVES.

THE ocular nerves are the oculo-motorius, trochlearis, and abducens; and since they are so closely related to each other, both in their functions and anatomical distribution, it will be convenient to form one group of the diseases to which they are liable. The subjoined diagram will suffice to remind the reader of the course and distribution of these nerves.

FIG. 32.

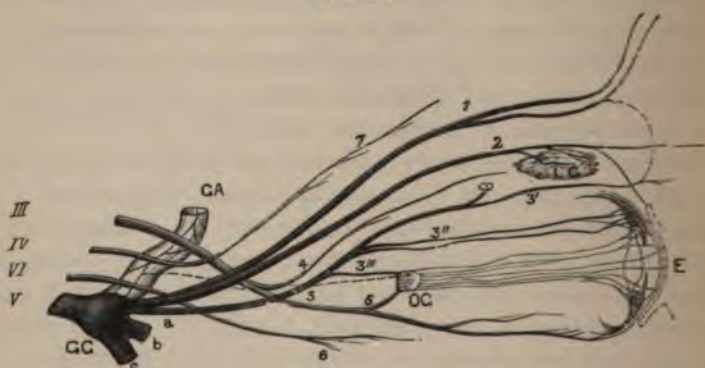


FIG. 32 (From Hermann's "Physiology"). *Diagram of the First or Ophthalmic Division of the Fifth, showing also the Third, Fourth, and Sixth Cranial Nerves.*

V, Sensory root of fifth nerve.

GG, Gasserian ganglion on larger root of the fifth nerve.

a, Ophthalmic division of the fifth nerve.

1, Frontal nerve. 2, Lachrymal nerve. 3, Nasal nerve.

LG, Lachrymal gland.

3', Infra-trochlear branch of nasal nerve.

3'', Long ciliary branches of nasal nerve.

3''', Branch of nasal nerve to ophthalmic ganglion.



§ 220. *Muscles of the Eyeball.*

The eyeball is moved by six muscles—the *recti inferior, superior, internus* and *externus*, and the *obliqui inferior* and *superior*. These muscles may be considered as three pairs, each pair rotating the eye round a particular axis. Each eye is capable of rotating round an immobile centre of rotation, its position being a little behind the centre of the globe, and where the horizontal and vertical axes intersect. The *primary position* of the eye is that which may be attained by looking at the distant horizon with the head vertical and the body upright. The visual axes are then parallel to one another, and to the median plane of the head. All other positions of the eye are called *secondary positions*. A vertical plane drawn through the centre of rotation at right angles to the primary visual axis is of importance, inasmuch as every change from the primary to a secondary position is brought about by a rotation of the eye round an axis lying in this plane (Listing). The chief axes in this plane are the transverse, rotation round which causes the eye to move up and down, and the vertical, rotation round which causes the eye to move from side to side. Rotation round other axes in this plane causes oblique movements.

The various movements of the eyeball may be arranged as follows (Foster):—

Straight Movements	{ Elevation.....	Rectus superior and obliquus inferior.
	{ Depression.....	Rectus inferior and obliquus superior.
	{ Adduction to nasal side.....	Rectus internus.
	{ Abduction to malar side.....	Rectus externus.
Oblique Movements	{ Elevation with adduction.....	Rectus superior and internus with obliquus inferior.
	{ Depression with adduction.....	Rectus inferior and internus with obliquus superior.
	{ Elevation with abduction.....	Rectus superior and externus with obliquus inferior.
	{ Depression with abduction.....	Rectus inferior and externus with obliquus superior.

§ 221. *Spasms of the Ocular Muscles.*

Various forms of tonic and clonic spasms may occur in the region of distribution of the ocular motor nerves. These spasms may be primarily subdivided into those which occur in the levator palpebræ superioris, the muscles of the eyeball, and the internal muscles of the eye.

OG, Ophthalmic ganglion.

*b*, Second division of fifth cut across. *c*, Third division of fifth cut across.

III, Third nerve (motorius oculi).

4, Upper division of third nerve.

5, Lower division of third nerve, near point where it gives the *short root* to the ophthalmic ganglion.

IV, Fourth nerve (n. trochlearis).

7, Its fibres passing to the *superior oblique*.

VI, Sixth nerve (n. abducens).

6, Its fibres passing to *external rectus*.

CA, Carotid artery.

E, Vertical section through anterior part of eyeball; conjunctiva indicated by dotted line.





after fright, or other emotional disturbances, are due to spasm of the internal rectus. Spasm of the internal rectus gives rise to homonymous diplopia, similar to that which occurs in paralysis of the sixth nerve; but paralysis of the sixth causes a fixed squint, while the power of moving the eye outward is lost or greatly impaired. In spasm of the internal rectus, on the other hand, there is a constant oscillation between the two images which alternately approach and recede from each other. All cases of spasmodic strabismus are accompanied by paroxysms of neuralgia, which extend over one-half of the head. It is often, also, associated with photophobia and lachrymation. Tonic spasm of the internal rectus may be caused by long-standing paralysis of its antagonist, the external rectus.

*Tonic spasm of the external rectus* is rare as an isolated affection; but Galezowski has observed it alternate with spasm of the internal rectus in a case of locomotor ataxy. Spasm of this muscle causes divergent squint, and gives rise to diplopia with crossed images; but contrary to what occurs in paralysis, the two images never remain fixed, but alternately approach and recede from each other.

The causes of spasmodic strabismus are generally the same as those which give rise to spasm of other muscles. It is necessary to examine with care for any source of irritation in the region of distribution of the fifth nerve, such as carious teeth. Exposure to cold, and especially exposure of one side of the face to a cold current of air, predisposes to this affection.

*Treatment.*—The treatment must be directed against the cause of the spasm whatever it may be, whether it be reflex, or direct irritation, or of central origin.

*Nystagmus* consists of a clonic spasm of the muscles of the eyeballs, giving rise to continual oscillatory or rotatory movements, which are entirely beyond the control of the patient. The ocular tremors sometimes consist of lateral oscillatory movements, when the external and internal recti are mainly implicated; while, at other times, the movements are rotatory, and then the oblique muscles are mainly affected. When the patient looks at a remote object, the trembling becomes very pronounced, and the eyes become more and more fixed in pro-

portion as they are directed to near or small objects. Nystagmus is always bilateral even when one eye is completely blind.

*Etiology.*—The causes of nystagmus are of two kinds, local and central. Amongst the local causes may be mentioned congenital defects of the optic nerves and retinae, pigmentary retinitis, congenital cataract, and corneal opacities.

Nystagmus of central origin appears to result, as a rule, from disease of one or other of the cerebellar peduncles. Friedreich has observed nystagmus in certain cases of locomotor ataxy, and in these it is probable that the lesion of the posterior columns had extended to the restiform bodies. This affection is often met with in insular sclerosis, and in these cases the diseased patches appear to be scattered about the cerebellum and its peduncles. Nystagmus may also arise in connection with basal meningitis, hydrocephalus, and other intracranial processes. It is often present in albinos, and colliers are frequently affected.

*Treatment.*—In many cases nystagmus does not produce any disturbance of vision, and consequently no interference is indicated. In other cases the disease is secondary, and the treatment must be directed against the primary affection. In cases of hypermetropia, myopia, or astigmatism, the use of appropriate glasses is indicated. If the trembling be due to spasm, or shortening of one or more of the ocular muscles, tenotomy must be had recourse to. In cases of more or less temporary nystagmus all sources of irritation, such as carious teeth, or diseases of remote organs, as the intestines and uterus, must be attended to.

*The sphincter of the iris*, which is innervated from the short root of the ophthalmic ciliary ganglion, may be affected both with clonic and tonic spasms. The clonic form of spasm of the iris is called *hippus*, and consists in quickly alternating contractions and relaxations of the sphincter. It is probable that the sympathetic nerve is also implicated in the disease. This condition, also called chorea of the iris, is sometimes manifested during the regressive period of paralysis of the third nerve.

*Tonic spasm of the sphincter of the iris* gives rise to contraction of the pupil, and is called *myosis spastica*. It is



usually a symptom of irritation of the oculo-motorius, either in its peripheral course or in its connection with the higher nerve centres.

It is consequently frequently associated with tonic contraction of one or more of the other muscles innervated by the third, and especially with tonic spasm of the internal rectus. It is not unfrequent as an isolated affection in those who have to make continuous efforts to see fine objects, and as a symptom of affections of the retina. It may also be produced artificially by various pharmaceutical means, as opium, nicotin, jaborandi, calabar bean, and its alkaloid eserine. Myosis is often a symptom of spinal disease and particularly of locomotor ataxy. Contraction of the pupil may also be due to paralysis of the sympathetic nerves of the iris.

*Treatment* must be directed to the cause of the myosis. Belladonna or atropine may be used to counteract the contraction of the pupil.

*Tonic spasm of the ciliary muscle* accommodates vision to near objects and renders the patient myopic. This condition is usually associated with symptoms of irritation in the region of distribution of the oculo-motorius, especially with *spastic myosis*. Spasm of accommodation may be artificially produced by various pharmaceutical agents, such as opium, calabar bean, and pilocarpin. Spasm of the ciliary muscle is rare as an idiopathic affection; but occurs sometimes in persons who make great efforts to see small objects, and Graefe has observed it as a reflex symptom of ophthalmic and facial neuralgia. Galezowski has observed it in a case of locomotor ataxy in a patient affected with myopia.

*Treatment* must be directed to the cause of the affection. Atropine, by paralysing the accommodation, is very useful, and concave glasses may be worn so as to correct the temporary condition of myopia.

#### § 222. *Paralysis of the Ocular Muscles.*

*General Remarks.*—Paralyses of the ocular muscles are not only of frequent occurrence, but they form most important symptoms in different cerebral and spinal diseases; hence, it is

very desirable that their presence should be detected and their varieties accurately determined.

*General Etiology.*—Paralysis of one or more of the ocular muscles occurs sometimes from exposure to cold, and is then supposed to be of *rheumatic* origin. The abducens and oculomotorius are the nerves most usually affected, and the paralysis is often limited to a few of their branches. The trochlear nerve is seldom affected.

The next most common causes of paralysees of these nerves are blows on the eyes, penetrating wounds of the orbit, and fractures of the skull. Another important cause is mechanical compression of the ocular nerves, either in the orbit or in their course along the base of the skull. Compression is produced by new formations, aneurisms, extravasations of blood, syphilitic affections, and basal meningitis. Neuritis arising either idiosyncratically, or in consequence of injury, also gives rise to paralysis. Paralysis of some of the ocular muscles is associated with bulbar paralysis and is frequently met with in locomotor ataxy.

*Syphilis* is a frequent cause of paralysis of the ocular muscles, and one of the most common nervous symptoms which occur in the later stages of this disease is paralysis of some of these muscles. Various anatomical changes may be the cause of the paralysis, as periostitis and exostoses of the orbit or base of the skull, or gummata at various points in the course of the nerves or in the brain.

*Diphtheritis* is also a frequent cause of paralysis of certain branches of the oculo-motorius. Ocular paralysis may also occur after other acute diseases, or exposure of the eye to strong impressions of light, excessive smoking, alcoholic abuse, and similar irregularities.

### § 223. *General Symptoms.*

*Diplopia.*—If, when the visual axis of one eye is directed to an object desired to be seen, the direction of the other deviates to some extent from that object, the image in the distorted eye falls on an eccentric portion of the retina, and two objects instead of one are seen. The image seen by the healthy eye forms on the macula, and consequently it is very distinct; while the



image seen by the distorted eye forms upon a more or less peripheric region of the retina, and is consequently faint and more or less confused; the former is therefore called the *true*, and the latter the *false*, image. This symptom is common to all forms of paralyses of the ocular muscles, and is called *binocular diplopia*. The more completely the muscle is paralysed the greater is the angle of deviation, and the more marked does the diplopia become.

*Neutralisation of the False Image.*—When the paralysis is of old date the patient learns to perceive objects only with the healthy eye, and the diplopia disappears; whilst the distorted eye, from long-continued disuse, suffers consecutive amblyopia. The diplopia, however, may be made to reappear by placing a coloured glass before the healthy eye, which differentiates the true image and permits that of the distorted eye to be perceived.

*Relations of the Images to one another.*—When one of the images is coloured, that of each eye is readily recognised. Assuming the left eye to be affected, the false image is displaced horizontally to the patient's right in outward squint (*Fig. 34, 1*), horizontally to the patient's left in inward squint (*Fig. 34, 2*); while the false is above the true image (*Fig. 34, 3 and 4*) in downward, and below it in upward squint (*Fig. 34, 5 and 6*). It will thus be seen that the false image assumes an opposite position to the distortion of the eye. In outward or inward squint the images are vertical and parallel with one another, but in all other forms of distortion the globe becomes rotated on its oblique axis by the unantagonised action of one or other of the obliqui muscles, and the false assumes an oblique position with reference to the true image, or, in other words, the false image is tilted (*Fig. 34, 3, 4, 5, and 6*). The false image is tilted in the opposite direction to the rotation of the eye. When, for instance, the superior oblique of the left eye is paralysed, the upper margin of the globe is rotated outwards to the left by the unantagonised action of the inferior oblique muscle, and the upper end of the false image is tilted to the patient's right (*Fig. 34, 6*).

The distance between the images becomes greater as the object is moved in the direction of the action of the paralysed muscles.

*Vertigo.*—Diplopia causes considerable embarrassment to the patient while walking. On ascending a stair, for instance, he sees two, and not knowing upon which to place his foot becomes confused and stumbles. Perception of distance is defective, so the patients become confused on endeavouring to grasp objects, This constant confusion causes great fatigue, vertigo, and sometimes vomiting. These symptoms disappear in great part when the distorted eye is closed.

*Compensatory Attitudes.*—The patient instinctively neutralises the perception of double images by placing the head in such an attitude that the paralysed muscle does not require to act. The attitude assumed by the head is necessarily different for each muscle paralysed.

*Secondary Deviation of the Sound Eye.*—This condition has already been described (§ 91).

*False Projection.*—When a person looks at an object with one eye only, a judgment of its position in space is formed by the sense of effort made to fix the object. If a patient with paralysis of the external rectus of the left eye be asked to touch an object coming before him from the left or paralysed side, he misses it by carrying his finger too far to the left, or to the side of the object corresponding to that of the paralysed muscle. The difficulty of immediately recognising the position of objects in space, called *false projection*, is caused by the fact that it is necessary to make an increased effort with the affected eye, so that the amount of rotation is over estimated; hence an erroneous judgment of the position of the body in space is formed.

*Secondary contraction of the antagonistic muscles* often occurs, which increases the extent of the deviation, and augments the distance of the double images from one another.

#### PARALYSIS OF PARTICULAR OCULAR MUSCLES.

§ 224. *Complete paralysis of the oculo-motorius* gives rise to drooping of the upper eyelid, or *ptosis*, the eyeball is drawn downwards and outwards by the action of the external rectus and superior oblique constituting divergent strabismus, dilatation and immobility of the pupil, and impairment of the power of accommodation. Every effort to move the eye in any



direction causes it to rotate downwards and outwards, and it gradually becomes fixed in this position by the secondary contraction of the external rectus and superior oblique. Double images appear almost over the whole field of vision, and fixation is only possible in a downward and outward direction. Secondary deviations of the healthy eye take place in all directions except in that towards the affected eye. Almost all the recti being paralysed, the eye is frequently protruded, giving rise to the appearance called *exophthalmos paralyticus*.

False projection of the field of vision occurs in every direction towards which futile efforts at fixation are made. The false image is to the patient's right when the left eye is affected, and when the object is in the horizontal position, the two images are on a level; but the false appears below the true image when the object is below, and above it when the object is above the horizontal line. The head is turned backwards and towards the healthy side, in a very oblique position.

§ 225. *Incomplete paralysis of the oculo-motorius* may affect one or several muscles.

(1) *Paralysis of the levator palpebræ superioris, or ptosis*, may occur as a separate affection, the result of injury, or spontaneously, but it is usually associated with paralysis of the superior rectus, which is also supplied by the superior branch of the oculo-motor nerve. The upper lid hangs motionless, the fissure of the lid is greatly narrowed, and when the eye is directed upwards the lid is not raised, and the horizontal fissures of the upper lid are effaced.

(2) *Paralysis of the superior rectus* gives rise to distortion, which is called *strabismus deorsum vergens*. The visual axis of the affected eye is directed more downwards than that of the sound eye, and the cornea also diverges a little outwards from the unopposed action of the inferior oblique. The false is seen above the true image, the vertical distance between the images increases according as the eyes are directed upwards and outwards, and the false image is tilted to the patient's right when the left eye is affected (*Fig. 34, 3*); and, provided there be no contraction of the inferior rectus, it disappears at the horizontal line. When the eyes are directed downwards, objects are seen

single, and the head is thrown back so as to counteract the paralysis.

(3) *Paralysis of the internal rectus* gives rise to *strabismus divergens*. The power of rotating the eye inwards is impaired or lost, and when the paralysis is complete the eye cannot be moved beyond the median line, and the axis of vision is inclined outwards. The images are on the same plane, vertical and parallel, the false one is to the patient's right when the left eye is affected (*Fig. 34, 1*), and the lateral distance between the images increases with the movement of the object towards the sound side. There is secondary deviation of the sound eye outwards, and in fixing an object the head is turned towards the healthy side.

(4) *Paralysis of the inferior rectus* causes squint, which is

FIG. 34.

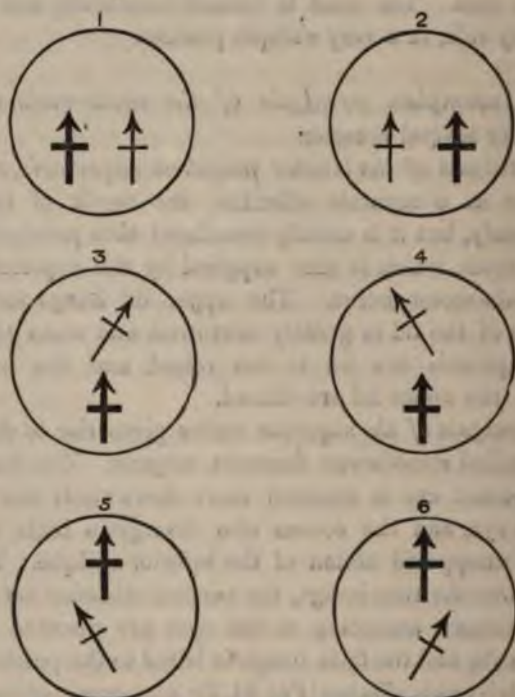


FIG. 34 (after Bristowe).—In the above diagram the thick cross represents the true image, the thin cross the false image. The left eye is supposed to be affected in all of them.



called *strabismus sursum vergens*. The symptoms are exactly the inverse of those caused by paralysis of the superior rectus. The affected eye is directed upwards, and slightly outwards. The false is below the true image, and tilted to the patient's left when the left eye is affected (*Fig. 34, 5*). Diplopia only occurs when the eye is directed downwards, and double vision is troublesome whenever the line of vision is lowered, as in walking and in all kinds of handiwork. When objects are held above the horizontal line the patient can see distinctly.

(5) *Paralysis of the inferior oblique* is rare as an isolated affection; and when it is associated with general paralysis of the third nerve its diagnosis is almost impossible. The affected eye is turned slightly downwards and inwards, double images are observed when the eyes are directed above the horizontal line, the false is above the true image, and tilted to the patient's left when the left eye is affected (*Fig. 34, 4*). The double images are scarcely perceptible in the horizontal line, and disappear when the eyes are directed downwards. According as the object is carried upwards and outwards the images become more and more separated, both vertically and laterally, and the tilting of the false image becomes more pronounced. On fixation in the median plane there is a false projection of the field of vision upwards and slightly outwards, and the head is thrown backwards, and the chin turned a little towards the healthy side, so that the lower and outer segment of the field of vision is chiefly brought into use. Ptosis and mydriasis frequently co-exist with paralysis of the inferior oblique.

(6) *Paralysis of the Sphincter Iridis (Mydriasis Paralytica)*. The pupil is in a medium state of dilatation, but it may be still further dilated with atropine. It is nearly or completely motionless when exposed to light, and does not contract when the eyes are strongly converged, or when efforts at accommodation are made. Vision is indistinct in consequence of circles of dispersion, bright light is disagreeable, and the power of accommodation is often, though not always, diminished.

(7) *Paralysis of the Ciliary Muscle (Cycloplegia)*.—This affection is often complicated with mydriasis, though it may occur as an independent affection. The patient, if not myopic, is unable to focus small objects, or read small print; and he forms an incorrect

estimate of the size and distance of small objects. This affection is very common in diphtheritic paralysis, and it may also accompany any other paralysis of the third nerve. In the latter paralysis the circular fibres of the iris are paralysed, and the dilators intact. It may result from disease in the short root of the ciliary ganglion, or in the trunk of the third nerve, or in the brain itself.

(8) *Paralytic myosis* is the condition of contraction of the pupil which occurs when the radiating fibres are paralysed, the circular ones remaining intact. It results from disease of the sympathetic root of the lenticular ganglion, or of the trunk of the sympathetic in the neck, or from disease or injury of the cilio-spinal region of the spinal cord.

(9) *Irido-plegia* is a term used to indicate total paralysis of the iris, both of its circular and radiating fibres. It is probably due to disease of the lenticular ganglion, and is then generally combined with cycloplegia (Hutchinson).

(10) *Ophthalmoplegia interna* is a name introduced by Mr. Hutchinson to indicate paralysis of all the muscles within the eyeball, these being the circular fibres and the dilators of the iris, and the ciliary muscle. This affection is probably due, as Mr. Hutchinson has pointed out, to disease of the lenticular ganglion. Of the eight cases communicated by Mr. Hutchinson, both eyes were affected in five. In three of the eight cases there was no history of syphilis; but the disease is generally of syphilitic origin.

(11) *The Argyll-Robertson symptom* consists in the absence of any contraction of the pupil on exposure of the eye to light, while movement with accommodation is normally retained. This symptom appears to be due to interruption of the reflex arc between the retina and the iris, and is consequently analogous to the absence of the patellar tendon reflex; both phenomena being generally observed in locomotor ataxia (Grainger Stewart, Wernicke, Hempel).

§ 226. *Paralysis of the Trochlear Nerve.*—The fourth or trochlear nerve supplies the superior oblique muscle, and it may be paralysed separately, especially in syphilis, or in connection with paralysis of the sixth nerve. Double images appear when



the eyes are directed downwards, and they become more and more separated both vertically and laterally, according as the object is carried downwards and outwards, while they become more closely approximated as the object is moved upwards. The double images are vertically superimposed, the false image being the lower of the two, and tilted to the patient's right when the left eye is affected (*Fig. 34, 6*). The image of the affected eye appears more remote than that of the healthy one. The secondary deviation is usually directed straight downwards. There is false projection of the field of vision downwards and a little outwards, and the head is inclined forwards and turned towards the healthy side. The feeling of giddiness is often well marked.

When antagonistic shortening of the inferior oblique exists, the deviation and diplopia extend more and more into the upper half of the field of vision.

§ 227. *Paralysis of the abducens nerve* causes *strabismus convergens*. The external rectus alone is paralysed in this affection. If the paralysis is complete, the eye cannot be rotated outwards beyond the middle line. Double images are seen when the eyes are turned in the horizontal line and to the paralysed side, the distance between them increases according as the object is moved to that side, the images are vertical, and the false one is to the left of the patient when the left eye is affected (*Fig. 34, 2*). Secondary deviation occurs towards the inner side, there is false projection of the field of vision towards the outer side, and the head is turned towards the affected side. The feeling of giddiness is severe, and may be accompanied by nausea and vomiting. Paralysis of the sixth nerve may occur as an isolated affection when due to rheumatism. It is sometimes bilateral in *tabes dorsalis*, or after acute cerebral meningitis.

*Course and Duration.*—Paralysis of one or more of the ocular muscles sometimes supervenes suddenly, appearing in the course of a night, as in the rheumatic and apoplectic varieties. At other times it develops more slowly and gradually, as in syphilis, neuritis, and chronic disease of the central nervous system. When the paralysis has reached a certain degree or become complete, the symptoms may remain stationary for a variable period; but after a time secondary contractures occur which

render the symptoms more marked, and may considerably retard or prevent complete recovery.

The duration of paralysis of the ocular muscles is very variable. Rheumatic paralyzes rarely last beyond a few weeks. Syphilitic paralyzes may last for months or years and yet recover, and the same is true of many central paralyzes. Many cases are of course quite incurable.

§ 228. *Diagnosis*.—In complicated cases, where several muscles of one or both eyes are affected, the diagnosis becomes difficult, and the reader must be referred to special treatises.

The nature of the primary lesion must be determined by a thorough investigation of all the circumstances of the case. In obscure cases care should be taken to examine closely for any trace of syphilis; while the connection between tabes dorsalis and ocular paralysis should not be forgotten.

With respect to the locality of the lesion, it must first be determined whether the paralysis is of centric or peripheral origin. In many cases ocular paralysis is accompanied by cerebral symptoms, as vertigo, sensory affections, hemiplegia, double optic neuritis, or by spinal symptoms, as ataxia; while at other times symptoms of some disease in the orbit or at the base of the cranium are present, and in all these cases it becomes comparatively easy to localise the lesion. The electrical test is not of much value in ocular paralysis, since it is impossible to stimulate the nerves and muscles without danger. The paralysis is often more complete when the lesion is peripheral than when it is central. The existence of symptoms resulting from implication of other nerves, and the history of the case, afford valuable aid in determining whether a peripheral lesion is situated at the base of the brain or in the orbit.

§ 229. *Prognosis*.—The prognosis greatly depends upon the cause of the affection. It is favourable in rheumatic cases, and even traumatic cases often recover. The prognosis is doubtful in syphilis, since many cases do not yield to treatment. The prognosis is good in the early stages of tabes dorsalis, since this symptom frequently disappears spontaneously, although it may recur. In central affections generally the prognosis



is grave. When galvanic treatment is followed by prompt improvement the prognosis is favourable.

§ 230. *Treatment*.—The cause of the paralysis must first be ascertained, and if possible removed, and more especially in cases of rheumatic or syphilitic origin. Electricity is the most important of the direct remedies, and the galvanic is preferable to the faradic current.

*Method of Galvanisation*.—The stable application of the current should be made transversely through the temples or through the mastoid processes, and also longitudinally from the eye to the neck. Galvanisation of the sympathetic appears to be of use at times. In order to act directly on the paralysed muscles the anode may be applied to the neck, and the cathode should glide over the closed eyelids, especially over those points which correspond to the paralysed muscles. The currents used should not be stronger than may be sufficient to produce distinct contractions of the facial muscles when the face is stroked with the cathode. The application should not exceed two or three minutes, and care should be taken to avoid interruptions and reversals. In favourable cases improvement begins very soon under galvanic treatment, although in some cases the treatment must be pursued many months before improvement occurs.

*Methods of Faradisation*.—Direct faradic stimulation in the vicinity of the attachments of the affected muscles may be adopted either by means of a small sponge electrode applied to the closed lids, or by means of a fine brush acting as an electrode and applied to the conjunctiva. Weak currents must be used, and for a short time only.

Of other remedies iodide of potassium is the most generally useful, since many cases of unknown origin, but which are not necessarily syphilitic, improve under its administration. Subcutaneous injection of strychnia has been found useful, and calabar bean may be employed in cases of mydriasis.

Gymnastics for the ocular muscles has been found of use in the slighter forms of the affection. These consist in exercising the enfeebled muscles by forced lateral movements, and by stereoscopic exercises with the view of suppressing the second image.

The fusion of the double images may be greatly aided by means of prisms. A pair of spectacles with one dull glass for the affected eye acts as a palliative remedy for the discomfort arising from the double images. If the affection resists all ordinary treatment, the propriety of resorting to operative procedure must be determined by the ophthalmic surgeon.

§ 231. *Progressive Paralysis of the Ocular Muscles.*

*Ophthalmoplegia Progressiva seu Externa.*—It has long been known that all, or almost all, of the ocular muscles on one or on both sides may become simultaneously affected with paralysis; but attention has been drawn by V. Graefe and others to a special form of this affection, which has been called progressive paralysis of the ocular muscles, an affection which runs a course parallel with progressive paralysis of the tongue and soft palate. This affection has also been carefully studied by Mr. Jonathan Hutchinson.\* The causes of the affection appear to be sometimes of a rheumatic nature; but probably a large proportion of the recorded cases were of syphilitic origin.

*Symptoms.*—Drooping of the eyelids, giving to the face a sleepy expression, is usually the first symptom; but all the muscles attached to the eyeball become gradually involved in the disease, and the movements of the globes become much restricted, or even wholly lost. The condition is usually bilateral, but the nerves are not always affected simultaneously, nor to the same degree, so that every variety of combination of paralysis of the ocular muscles may be observed.

The affection is often associated with paroxysms of lancinating pains in the head, dizziness, and febrile symptoms; while blindness, with white atrophy of the optic nerves was present in one-third of the cases observed by Mr. Jonathan Hutchinson. The trigeminal and facial nerves occasionally participate in the paralysis, although implication of the facial nerves is very rare. The affection is sometimes accompanied by feebleness of the lower extremities, neuralgic pains, and other symptoms of locomotor ataxy.

\* Medico-Chirurgical Transactions, Vol. LXII., 1879, p. 307.



*Morbid Anatomy.*—In one of the cases recorded by Mr. J. Hutchinson, Dr. Gowers found degeneration of the roots of the ocular nerves, and disappearance of the cells from their centre of origin. The changes observed resembled those found in progressive muscular atrophy, and, indeed, the two affections are closely allied clinically. Ophthalmoplegia externa is at times a part of progressive locomotor ataxy (Hutchinson).

*Prognosis.*—The prognosis is much the same as in affections of other nerves, and, on the whole, it is not unfavourable.

*Treatment.*—The treatment is the same as when these nerves are separately affected. If any trace of syphilis be detected, iodide of potassium must be administered, and this drug is useful in other cases, as, for example, when the disease is the result of basal rheumatic periostitis, or chronic meningitis.

#### (II.)—DISEASES OF THE FACIAL NERVE.

The facial nerve is the motor nerve of the face. It is distributed to most of the muscles of the ear and to the muscles of the scalp, to those of the mouth, nose, and eyelids, and to the cutaneous muscles of the neck. The annexed diagrams will remind the reader of the course and distribution of the facial nerve, and the muscles to which it is supplied, without detailed description.

#### § 232. *Spasm in the Area of Distribution of the Facial Nerve.*

*Histrionic Spasm of the Face. Mimic Convulsion. Convulsive Tic.*

Spasmodic action of the facial muscles occurs in various diseases of the nervous system, as epilepsy and tetanus; but the disease under consideration at present is a local affection confined to the muscles supplied by one of the seventh pair of nerves, or by one of its branches. The disease sometimes arises in the absence of any recognisable cause. Occasionally it appears to be inherited. Rosenthal mentions the case of a family where a mother, her son, sister, and two other relations, were affected with a more or less degree of facial spasm. The affection often results from emotional disturbances. In the case of a lady known to me, the spasm began in consequence

of a long and anxious nursing of a sick relative; but it is possible that general exhaustion and exposure to a draught of cold air had concurred to cause the disease in this case. Romberg mentions an instance in which the disease affected a female in consequence of the shock caused by the sudden death of her husband.

FIG. 35.

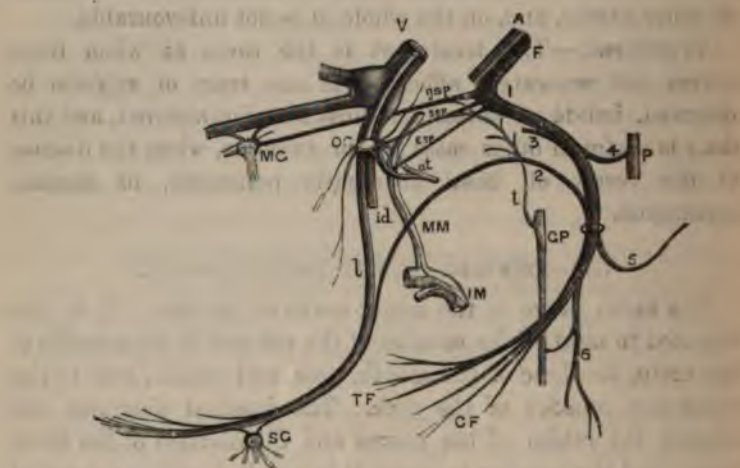


FIG. 35 (From Hermann's "Physiology"). *Diagram of the Facial Nerve, its connections and branches.*

F, The facial nerve.

A, Auditory nerve.

1, The geniculate ganglion.

*gsp*, Great superficial petrosal nerve connecting the facial and Meckel's ganglion.

*ssp*, Small superficial petrosal nerve connecting the facial with the Otic ganglion and with the tympanic branch of the glosso-pharyngeal.

*esp*, External superficial petrosal connecting the facial with the plexus on the middle meningeal artery.

2, Chorda tympani, joining lingual nerve.

3, Nerve to stapedius muscle.

4, Communicating branch with the ganglion of the root of the vagus.

5, Posterior auricular nerve.

6, Branch to the stylo-hyoid and digastric muscles.

TF, Temporo-facial division } to muscles of expression.

CF, Cervico-facial division }

V, Fifth nerve. *at*, Auriculo-temporal branch.

*id*, Inferior dental nerve.

*l*, Lingual nerve.

MG, Meckel's ganglion.

OG, Otic ganglion.

SG, Submaxillary ganglion.

IM, Internal maxillary artery.

MM, Middle meningeal artery.

P, Pneumogastric nerve.

GP, Glosso-pharyngeal nerve.

*t*, Its tympanic branch (nerve of Jacobson).



The disease is often caused either by direct or reflex irritation of the facial nerve. The direct causes are the same generally as those which cause facial paralysis, such as exposure to cold, tumour at the base of the brain (Schuh), inflamed gland in the vicinity of the stylo-mastoid foramen (Romberg), abscess of the parotid gland (Debrou), caries of the petrous portion of the temporal bone (Oppolzer), and otitis (Rosenthal, Remak). The reflex irritation generally takes place through the trigeminus; the most usual sources of irritation being trigeminal neuralgia, carious teeth, irritation of the eyeball and of the conjunctiva. The source of the irritation is sometimes remote. Remak relates a case of spasmodic actions of the muscles of the hand and arm extending to the muscles of the neck and face of the same side, which was caused by neuritis of the cervico-brachialis, with knotty points in the course of the nerve. The disease

FIG. 36.



FIG. 36 (From Heath's "Anatomy"). *Muscles of the Head and Face.*—1, Frontal portion of the occipito-frontalis. 2, Its occipital portion. 3, Its aponeurosis. 4, Orbicularis palpebrarum, which conceals the corrugator supercilii and tensor tarsi. 5, Pyramidalis nasi. 6, Compressor nasii. 7, Orbicularis oris. 8, Levator labii superioris alaeque nasi; the adjoining fasciculus between numbers 8 and 9 is the labial portion of the muscle. 9, Levator labii superioris proprius; the lower part of the levator anguli oris is seen between the muscles 10 and 11. 10, Zygomaticus minor. 11, Zygomaticus major. 12, Depressor labii inferioris. 13, Depressor anguli oris. 14, Levator labii inferioris. 15, Superficial portion of the masseter. 16, Part of its deep portion. 17, Attrahens aurem. 18, Buccinator. 19, Attollens aurem. 20, Temporal fascia covering the temporal muscle. 21, Retrahens aurem. 22, Anterior belly of the digastricus. 23, Stylo-hyoid pierced by posterior belly of the digastric. 24, Mylo-hyoideus. 25, Sterno-mastoid. 26, Trapezius.

may also be caused, especially in children, by intestinal irritation from the presence of worms, and in females by uterine irritation.

§ 233. *Symptoms.*—Histrionic spasm of the face may be *clonic* or *tonic*, the former variety being by far the more frequent of the two. In the clonic form the attacks come on in paroxysms, consisting of sudden and violent contractions and relaxations of some of the muscles of the face. The duration of each paroxysm varies from a few seconds to a few minutes, and then ceases, only to be followed after a short interval by another. The spasm is generally confined to the one side of the face, so that the contortions and grimaces of the affected side contrast strangely with the calm and natural expression of the opposite side. The contortions produced are extremely variable, and consist mainly of elevation and depression of the occipital and frontal muscles, corrugation of the eyebrows, twitching or winking of the eyelids, elevation of the cheek and of the nostrils, and distortion of the angle of the mouth; but these may be present in every imaginable combination. The spasm may be partial, being limited to the muscles supplied by single branches of the nerve. The palpebral twigs alone are sometimes affected. In the tonic form the eyelids are firmly closed, constituting *blepharospasm*, and in the clonic variety the affection manifests itself by a rapid winking, hence called *nictitating spasm*; while at other times there may be only slight twitching movements of one of the eyelids, generally the lower. When the malar and labial branches are affected it gives rise to a convulsive grin, resembling laughing, on one or on both sides, and hence called the sardonic laugh, or *risus caninus*, or cynic spasm.

The whole of the facial nerve may at times be implicated, so that not only the facial muscles but the platysma myoides may be affected with spasm. The affection also occurs in connection with spasm of the muscles supplied by other nerves, as the fifth, hypoglossal, spinal accessory, or some of the spinal nerves.

The muscles supplied by the auriculo-muscular branches of the facial are rarely affected. Romberg mentions two cases in which these muscles were the subjects of spasm. In the first



case the spasmodic movements occurred in paroxysms, and appeared after partial recovery from apoplexy accompanied by paralysis of the right arm; and in the other case the spasm of the ears preceded an epileptic seizure, and thus, as Romberg remarks, took the place of the aura. It is quite evident, therefore, that neither of these cases is a true instance of the disease under consideration at present.

The paroxysms of facial spasm are of variable duration, and may be excited by emotional disturbances, voluntary movements of the affected muscles, reflex irritation, and in severe cases the spasm supervenes spontaneously and without any exciting cause. The paroxysms are not usually accompanied by pain, although occasionally at an early stage a slight degree of pain or numbness may be present, and the patient may also complain of headache, *tinnitus aurium*; but no kind of disorder of sensibility ever forms a prominent feature of the disease. The disease is not accompanied by any paralysis. All the voluntary movements can be executed, and there are no vaso-motor nor secretory disturbances, and the electrical reactions are normal. Certain points may be found, pressure on which will arrest the spasm. These points when present correspond with the pressure points in trifacial neuralgia. The paroxysm may continue, but as a rule disappears during sleep.

*The tonic form* of facial spasm was first clearly recognised by Dr. Marshall Hall. In this form the muscular contraction is persistent. The tip of the nose, angle of the mouth, and the chin are drawn to the opposite side, and the furrows and dimples of that side are rendered deeper. The contracted muscles render the tissues on the affected side too scanty for covering the orifices, so that the eye on the affected side is closed, a tightness is induced at the angle of the mouth, and when the angle of the mouth is moved from the eye, as in speaking, it is difficult to keep the eye closed. Articulation is rendered indistinct, and the bolus of food tends to collect between the teeth and the affected cheek, causing slight trouble during mastication; but there is no difficulty of deglutition.

*Blepharospasm* is the most important of the partial forms of facial spasm. This form of the affection has been carefully studied by Von Graefe. The eyelids are firmly closed in paroxysms,

each of which may last from a few minutes to a few hours, but may occasionally extend over a period of weeks or of months. Blepharospasm is generally accompanied by photophobia, and it is sometimes associated with trigeminal neuralgia. Pressure on the points which correspond to the tender points in trigeminal neuralgia, and especially over the infra-orbital foramen, may completely arrest the spasm. Blepharospasm is generally of reflex origin, and induced by irritation of the fifth, and more especially of its ocular branches.

§ 234. *Course, Duration, Terminations, and Prognosis.*—The course of the disease is generally chronic, and it may last during the lifetime of the patient. Complete recovery is probably the rarest termination, the most frequent being partial recovery with relapses. The prognosis varies with the cause. It is most favourable when the spasm depends upon a curable disease of the eye or conjunctiva, or upon some other source of peripheral irritation.

§ 235. *Diagnosis.*—The tonic form of facial spasm may be mistaken for facial paralysis of the opposite side, but in the former affection the immobility is found on the side to which the face is drawn, while the reverse obtains in the latter. In spasm the affected muscles are prominent and rigid, and their faradic contractility is normal or exaggerated; while in paralysis the affected muscles are soft, flaccid, and their faradic contractility is diminished or lost.

§ 236. *Morbid Anatomy and Pathology.*—The disease is no doubt caused by irritation, either direct or reflex, of the seventh nerve, or of one of its branches. The irritation may be caused by disease, such as a tumour or cicatrix in the vicinity of the nerve at any part of its course. The affection is at other times caused in a reflex manner by irritation of the fifth.

§ 237. *Treatment.*—The first aim of treatment must be to remove the cause of the affection. If the disease is the result of exposure to cold, soothing applications, such as the vapour bath, ought to be applied, while opium may be given in some form



internally, or a subcutaneous injection of morphia may be administered. When it is caused by irritation of the facial nerve by a tumour, enlarged glands, or cicatrix, the source of the irritation must be removed if possible. When reflex irritation exists, the treatment must be directed against its source. The irritation sometimes originates in the intestinal canal, especially from the presence of worms, or from the uterus; and the functions of these organs must be carefully investigated and regulated.

The cause of the irritation may be conveyed to the nerve through the blood; hence the constitutional state of the patient must be investigated. If rheumatism be present, salicylic acid would be a suitable remedy, and, if that failed, iodide of potassium. If the patient be scrofulous, sub-carbonate of iron and cod-liver oil would be likely to prove the best remedies.

If the affection is associated with chorea, arsenic and bromide of zinc are the most likely remedies to succeed, or large doses of the bromide of potassium might be tried; but I have much more faith in arsenic given in progressively increasing doses up to from seven to ten minims of the liquor than in any other remedy.

The application of chloroform, ether spray, and subcutaneous injections of morphia will be found useful alternately as local applications. Continued pressure by means of a small compress, either on the trunk of the nerve, at its exit from the stylo-mastoid foramen, or on one of its divisions, according to the extent of the disease, has been found useful. When a point can be discovered over one of the branches of the fifth nerve, pressure upon which arrests the spasm, the compress ought to be applied over this point. Dieffenbach divided subcutaneously all the muscles affected with spasm, and the convulsions immediately ceased. A year and a half after the operation, all that remained was some degree of tremor and agitation. Section of the facial nerve has been recommended, but the paralysis which results is a fatal objection to the treatment. Nerve stretching might probably be found useful.

The best direct treatment consists of the application of the constant current. When points of arrests are obtained, the anode should be placed over one of these; while in other cases

the descending current may be passed along the facial nerve to the affected muscles.

The patient should be directed to exert his will to the utmost to control the facial movements; just as he would do in endeavouring to overcome a bad habit.

§ 238. *Paralysis of the Seventh Nerve. Mimetic Facial Paralysis. Hemiplegia and Diplegia Facialis. Prosopalgia. Bell's Paralysis.*

Paralysis of the facial nerve was first thoroughly investigated by Sir Charles Bell; hence it is now frequently called *Bell's Paralysis*. There is probably no nerve in the body so liable to be independently attacked as the facial. Its superficial position exposes it to various traumatic influences; while its course through a long and narrow bony canal, and its proximity to organs very liable to be diseased, afford numerous opportunities for the occurrence of various lesions of the nerve, and render it peculiarly liable to become secondarily implicated.

Paralysis of the facial is sometimes unilateral, sometimes bilateral, and all the branches, or particular branches only, may be affected.

§ 239. *Etiology.*—Exposure of one side to cold is one of the most frequent causes of paralysis of the facial, and it is then called rheumatic paralysis. The paralysis is probably due to a slight neuritis, followed by serous or plastic exudation into the sheath of the nerve, compressing the nerve fibres. The neuritis may occur either in the canal or external to it. When the latter portion is affected, the compression produced is not sufficiently great to cause degeneration of the nerve fibres, so that simple paralysis results; but when the nerve is affected within the canal, the fibres undergo strong compression, and consequently degenerate, so that a severe paralysis with the reaction of degeneration results. Some persons appear to manifest a predisposition to this form of paralysis. Eulenburg mentions the case of a young man under his care, who suffered twice from right-sided, and thrice from left-sided facial paralysis of a very obstinate character.

Facial paralysis occurs at all ages, but it is most frequent



between twenty and forty years of age. Sex does not seem to exert any influence, and each side of the face is about equally liable to be affected. Facial paralysis is frequently the result of injury. It has been observed to follow a severe blow on the ear, gunshot and various other wounds, fractures of the skull and of the temporal bone, and wounds caused by missiles entering the internal ear. It also occurs after extirpation of the parotid gland and other surgical operations about the face and ear; and it may be caused in new-born infants by the pressure of the forceps (Duchenne). Affections of the parotid and of the neighbouring parts may cause facial paralysis either by pressure on the trunk of the nerve or of one of its branches, or by extension of the morbid process to it. Amongst the lesions which cause paralysis may be mentioned swelled lymphatic glands; abscess or infiltration of the cellular tissue in the neighbourhood of the stylo-mastoid foramen; inflammation, abscess, and tumours of the parotid gland; deep-seated ulcerations and cicatrices, such as occur after scrofulous abscesses of the glands.

Various diseases of the ear may also cause facial paralysis, and of all the causes of this affection suppurative *otitis interna*, followed by destructive changes in the temporal bone, is the most frequent. Bony tumours and neoplastic formations of all kinds proceeding from the internal ear may also lead to compression or destruction of the nerve. Facial paralysis, as an isolated affection, is rare as a sequel after acute diseases such as diphtheria and variola, unless indeed there is disease of the temporal bone; but it frequently occurs in the course of syphilis. Syphilitic periostitis, meningitis, exostoses, and gummata at the base of the skull, in the temporal bone, or in the brain, very frequently implicate the facial nerve.

Facial paralysis occurs as a symptom of various intracranial diseases; and the following groups may be distinguished:—(1) *Basal Paralysis*, caused by disease or compression of the nerves at the base of the skull or in its passage through the medulla to its nucleus; (2) Disease of the nucleus itself, or in its neighbourhood; (3) Lesion of the fibres which connect the facial nucleus with the cortex of the opposite hemisphere; and (4) disease of the motor centre in the cortex situated in the posterior part of the second frontal convolution.

Facial paralysis is rare as a symptom of disease of the spinal cord, but it may occur when the disease progresses as far as the upper end of the medulla. The facial nucleus may, for instance, be implicated in acute ascending paralysis of the cord, and it may also occur in *tabes dorsalis*.

§ 240. *Symptoms*.—The onset of facial paralysis differs according to its cause. It appears suddenly when it results from a traumatic lesion of the nerve, and when it results from exposure to cold the patient is usually surprised to find one side of his face paralysed in the morning. When the paralysis results from disease which invades the nerve secondarily, either by gradual compression or by altering its texture, the paralytic symptoms become slowly and gradually developed, and spread from branch to branch of the nerve. Premonitory symptoms may be experienced for some days before the appearance of the paralysis, consisting of pain on the side of the face which is subsequently paralysed, noises in the ear, deafness, and abnormal sensations of taste on the same side.

The symptoms of complete unilateral facial paralysis are very characteristic. The paralysed side of the face loses its wrinkles and furrows; it appears smooth, flaccid, and expressionless. From the loss of muscular tone the paralysed side falls to a lower level than the healthy one, and this distortion is much increased when the facial muscles are called into action during smiling and talking. The patient cannot wrinkle his forehead nor elevate his eyebrow; he cannot close his eye, and when he attempts to do so the eyeball is observed to roll upwards and inwards, or occasionally upwards and outwards. Owing to paralysis of Horner's muscle, the tears cannot enter the lachrymal canal, and therefore flow over the cheek. The power of winking is lost, and the eye remains open during sleep (*lagophthalmos*), and being no longer protected from the contact of foreign particles, it often becomes irritated and inflamed. The nostril on the paralysed side instead of expanding during inspiration falls in, and the tip of the nose is sometimes drawn to the healthy side. The mouth is also drawn obliquely over to the healthy side, and the distortion becomes more pronounced during all mimetic movements, as in crying, laughing, and speaking. Paralysis of



the buccinator causes the cheek to puff out in speaking and other expiratory actions, the pronunciation of the labial consonants is impaired, attempts at blowing or whistling fail, the air escaping through the paralysed fissure of the lips, the saliva dribbles from the affected side, and the food is apt to accumulate between the inner surface of the cheek and the teeth.

The external muscles of the ear are also paralysed, but since, in the majority of people, these muscles are not under voluntary control, impairment of movement in them is not readily detected. Paralysis of the platysma, of the posterior belly of the digastric, and of the stylo-hyoid muscles can sometimes be demonstrated.

If the lesion of the nerve is situated above the geniculate ganglion, the levator palati and azygos uvulæ become paralysed, and consequently the soft palate on the side corresponding to the facial paralysis hangs loosely downwards, occupies a lower position than on the sound side, and manifests diminished action on reflex irritation and in phonation. The uvula is also distorted, being curved to one side and somewhat forwards, the tip being sometimes directed towards and sometimes away from the paralysed side (Sanders). This condition will be discussed more at length when the various forms of paralysis of the muscles of the soft palate are under consideration.

If the facial nerve is diseased above the point where the branch to the stapedius is given off (*Fig. 35, 3*) that muscle becomes paralysed, and the membrana tympani is rendered unduly tense by the over-action of the tensor tympani muscle.

The researches of Lucæ\* have shown that this condition causes an abnormal acuteness of hearing of all musical tones, or sometimes an abnormal power of perceiving deep notes, often accompanied by a subjective sound of high pitch. Roux, speaking of an attack of facial paralysis, from which he himself suffered, mentions a painful vibration of the tympanic membrane on the affected side for moderately strong sounds. If, therefore, abnormal acuteness of hearing accompanies facial paralysis, it may be inferred that the facial nerve is affected above the point where the little branch for the stapedius is given off.

\* Berlin Klin Wochenschrift, 1874, Nos. 14, 16, 17.

It is scarcely necessary to add that the auditory and facial nerves may be simultaneously affected by diseases at the base of the brain, in the meatus auditorius internus, and of the middle ear and the adjoining parts of the temporal bone. When that is the case, the facial paralysis is accompanied by complete deafness of the ear on the affected side.

§ 241. *The chorda tympani* contains three kinds of fibres : (1) Fibres which minister to the sense of taste for the anterior two-thirds of the tongue ; (2) secretory fibres for the submaxillary gland ; (3) fibres distributed to the vessels of the gland. The first kind, or the gustatory fibres, accompany the facial only for a short part of its course, leaving it probably at the level of the geniculate ganglion by the great superficial petrosal nerve (Schiff). If, therefore, the lesion is situated anywhere between the geniculate ganglion and the point of origin of the chorda tympani from the facial, the sense of taste in the anterior two-thirds of the tongue will be abolished ; while taste will remain unaffected when the lesion is situated at the base of the brain above the ganglion, or below the point where the chorda tympani leaves the facial (*Fig. 35, 2*).

The disorder of taste consists of diminution or abolition of the sense of taste for acid, sweet, or saline substances in the anterior two-thirds of the tongue on the affected side. Disease of the secretory fibres causes diminution of the salivary secretion on the paralysed side, the patients often complaining of an abnormal dryness of that side of the mouth.

Diminution of the sense of smell has occasionally been observed on the paralysed side. This is caused partly by dryness of the nostril on account of the insufficient discharge of tears ; and partly because the paralysis of the levator alæ nasi and the compressor naris interferes with the access of air to the olfactory chambers.

In all cases of isolated paralysis of the facial the tongue lies straight on the floor of the mouth, and does not deviate when protruded. There is no paralysis of the tongue, but an apparent deviation on protrusion occurs, because the affected angle of the mouth is drawn over to the healthy side.

The sensibility of the paralysed side of the face is generally



normal. When, however, the peripheral divisions of the nerve are affected, branches of the fifth are usually implicated in the disease, and there will be corresponding disturbances of sensibility.

*The reflex movements* are abolished in the paralysed muscles in all cases of peripheral origin; but in paralysis of central origin, more especially when the disease is situated in the hemispheres, reflex irritability is completely preserved.

*Associated movements* are likewise abolished in peripheral and preserved in central paralysis.

*The electrical reactions* of the paralysed nerves and muscles have been carefully studied in the form which has been called rheumatic facial paralysis.

Erb distinguishes three groups, with respect to the electrical reactions obtained:—

1. The first group comprises those cases in which the reaction to both the galvanic and faradic currents of the nerve and muscles is entirely unaffected. The prognosis is extremely favourable in these cases, recovery taking place in two or three weeks.

2. The second group comprises all the cases which manifest the "reaction of degeneration," and the prognosis of this group is essentially unfavourable. The affection may be expected to last from two, four, six months, or even longer; and traces of the paralysis, consisting of a certain stiffness of the movements, slight contractures, and muscular twitchings, are often observable for years.

3. The third comprises all the transitional forms between the first two groups. The prognosis is relatively favourable in this group, recovery taking place in from four to six weeks. In such cases the faradic and galvanic excitability of the nerve may be diminished as early as the close of the first week, but is never entirely lost. The muscles exhibit increase and qualitative alteration of the galvanic excitability, as well as increase of mechanical excitability. The characteristic feature of this group is, therefore, that the muscles exhibit the reaction of degeneration at a time when the electrical reaction of the nerve shows that it has not undergone complete degeneration.

*In Traumatic Paralysis* of the facial nerve, and in that which results from severe compression, such as is caused by tumours at the base of the brain, the reaction of degeneration appears in its typical form. In paralysis, from disease in or near the facial nucleus, as in bulbar paralysis, a simple and moderate diminution of the electric excitability occurs in the nerve and muscles when the paralysis has lasted for some time.

In paralysis from lesion above the pons, the electric excitability is usually unaltered; but occasionally a slight increase may be observed.

§ 242. *Diplegia Facialis*, or bilateral facial paralysis, deserves special mention. A case of this affection was recorded by Sir Charles Bell, in 1836, and Davaine wrote an important monograph on the subject in 1852; and the disease has been studied since that time by Wachsmuth, Pierreson, and others.

The disease often results from a simultaneous lesion, such as compression by a tumour of both facial nerves, when they are close together as at the base of the cranium, or in their course through the medulla oblongata and pons. It is, however, most frequently observed in connection with progressive bulbar paralysis. A curious case has been recorded by Romberg and Magnus, in which the patient had two attacks of left hemiplegia, with aphasia and bilateral facial paralysis. At the post-mortem a hæmorrhagic cyst was found "at the external edge of the right hemisphere of the cerebrum, where the anterior lobe adjoins the middle one." The lesion was evidently situated in the posterior part of the second and third frontal convolutions of the right hemisphere; so that the cortical centres for the movements of the opposite side of the face and of the tongue were destroyed. The remarkable feature of this case was, not that aphasia was associated with left hemiplegia, which is not unusual, but that the mimetic movements of both sides of the face were governed from one hemisphere of the brain. Bilateral paralysis of the facial has been observed to arise from exposure to cold, from wounds affecting both temporal bones, from bilateral *otitis interna*, and from caries of the temporal bones.

*Symptoms.*—In facial diplegia the immobility which is present on one side in the unilateral affection now appears on both sides; but the oblique position of the chin, mouth, and nose is absent. The face is smooth, fixed, and expressionless, even when the emotions are powerfully excited, and the patient, in the apt language of Romberg, "laughs and cries as from behind a mask." When the lesion implicates all the branches of both nerves the patient is unable to wink or close either eye, and the tears flow over both cheeks. Both cheeks are flaccid and puff out during expiratory acts; mastication is imperfectly performed, owing to the food lodging between the teeth and cheeks; some difficulty is also experienced in swallowing, due to paralysis of the soft palate and of the stylo-hyoid and digastric muscles; and the



voice has a nasal tone, and fluids readily escape through the nose, during attempts at deglutition, from paralysis of the soft palate. The nostrils fall in during inspiration, causing a considerable amount of discomfort and difficulty in breathing. The influence of the facial nerves on respiration is best exemplified in animals like the horse, with very mobile nares, and in which section of both facials has been known to have caused asphyxia. Articulation is greatly impaired, as manifested by inability to pronounce the vowels "o" and "u" and the labial consonants. Taste is abolished in some cases in the anterior two-thirds of the tongue, and the sense of hearing may also be affected from paralysis of the stapedii muscles, without the auditory nerve being implicated in the disease.

§ 243. *Course, Duration, and Terminations.*—The course of facial paralysis varies greatly, according to its cause. In paralysis of rheumatic origin the prognosis is greatly aided by the electrical reactions already described.

*Secondary contracture* is apt to occur in the paralysed muscles in the course of the third or fourth month. It begins as a slight tonic contraction, generally beginning at the angle of the mouth, which is drawn upwards and outwards to the affected side, and remains fixed in that position. The nasolabial fold again becomes distinctly marked, the cheek becomes tense and firmly pressed against the teeth, the orbicularis palpebrarum may be affected, making the fissure of the eyelid narrower, so that the face assumes a very peculiar and characteristic aspect.

*Spontaneous movements* may occur in the muscles during recovery. These movements consist of twitching of some of the muscles, which may sometimes be so intense as to simulate convulsive tic.

These contractions occur sometimes when the patient is quiescent, while at other times they appear as associated movements. When an attempt is made to close the eye, for instance, the angle of the mouth is drawn outwards and upwards; and, conversely, when an attempt is made to draw the angle of the mouth to one side, the fissure of the lid contracts, while the zygomatici contract on attempting to elevate the eyebrow.

Contractions may also occur in a reflex manner, either through the fifth by touching or pricking the skin, or touching the eyelashes, or through the optic nerve by making a rapid movement towards the eye.

Traumatic paralysees are, as a rule, very protracted. In favourable cases recovery takes place in the course of four to six months; but it is frequently very incomplete, and generally the paralysis remains for life.

In paralysis from pressure, and in other peripheral paralysees, such as those resulting from otitis interna, neuritis, and syphilis, the course of the affection varies with the nature of the lesion and the severity of the case. Electrical examination is, as usual, useful in prognosis.

§ 244. *Diagnosis.*—The diagnosis presents no difficulty except in the slighter forms of the affection. In these, want of symmetry may be detected in the action of the muscles of the two sides by careful attention to the play of the features under varying emotions, and by testing the finer and more complicated movements, such as those engaged in pronouncing difficult words, whistling, showing the teeth, and various other actions. Considerable difficulty is experienced in diagnosing facial paralysis in infants; the principal signs to be depended on being the distortion that occurs on crying, the difficulty of sucking, and the lagophthalmos during sleep.

Erb lays down very careful and precise rules for the diagnosis of the seat of the lesion, of which the following is a summary:—

*Peripheral Facial Paralysis.*—Implication of all the external branches of the nerve, lagophthalmos especially continuing during sleep, atrophy of the paralysed muscles, the presence of the reaction of degeneration, absence of reflex and associated movements, the presence of external wounds or other injury in the vicinity of the nerve, disease of the organs adjoining the peripheral part of the nerve, as of the parotid gland, the internal ear, the temporal bone, the other nerves at the base of the brain, and the absence of all cerebral symptoms.

*Cerebral Facial Paralysis.*—The branches of the nerve distributed to the upper part of the face not being implicated in the disease, the fact that the eye can be closed voluntarily and during sleep, the preservation of reflex acts, the absence of atrophy of the muscles and of the reaction of degeneration, and the presence of other cerebral symptoms, as vertigo,



sensory disturbances, hemiplegia, weakness of the tongue, disorders of speech, and considerable difficulty in swallowing.

Each of these main divisions may be subdivided into several groups.

*A.—Peripheral Paralysis.*

1. If the lesion be situated external to the Fallopian canal, the muscles of the face are alone paralysed.

2. If the lesion be situated in the Fallopian canal, but below the point at which the chorda tympani is given off, the muscles of the external ear are paralysed in addition to those of the face.

3. If the lesion be situated between the point at which the chorda tympani is given off and the point of origin of the small branch to the stapedius, there are, in addition to the symptoms already mentioned, abolition of taste on the anterior two-thirds of the tongue and diminution of the salivary secretion of the affected side.

4. If the lesion be situated between the point of origin of the nerve to the stapedius and the geniculate ganglion, the same symptoms are present along with abnormal acuteness of hearing.

5. If the geniculate ganglion itself is diseased, all the previous signs are present, and in addition paralysis of the soft palate and distortion of the uvula.

6. If the lesion is situated in the nerve above the geniculate ganglion, all the previous signs are present with the exception of the disorder of the sense of taste. The auditory nerve is also frequently implicated, and then there is dulness of hearing on the affected side.

*B.—Intracranial Paralysis.*

1. Lesion of the facial nucleus gives rise to complete paralysis of the facial branches, paresis of the velum palati, simple diminution of electrical excitability; but there are no gustatory disturbances. That the lesion is situated in the medulla is rendered more certain by the hypoglossus, spinal accessory, vagus, trigeminus, or the abducens being affected.

2. When the lesion is situated in the lower and middle regions of the pons there is complete paralysis of the facial branches, paresis of the velum palati, and paralysis of the extremities on the opposite side of the body, or *alternate* paralysis. When the lesion is situated in the upper part of the pons, above the decussation of the fibres which connect the facial nuclei with the posterior part of the second frontal convolutions of the cortex, the paralysis of the extremities is on the same side as that of the face. Diseases of the pons may exert pressure on the facial nerves at the base of the brain, and thus induce some of the symptoms of peripheral paralysis of the nerve.

3. When the disease is situated above the pons in one of the crura or hemispheres there is only paralysis of the lower facial branches, paresis of

the velum palati, and paralysis of the extremities on the same side. Lesion of the crus gives rise to facial paralysis and crossed paralysis of the third nerve.

The diagnosis of the nature of the lesion must be based on general pathological grounds.

§ 245. *Prognosis*.—The prognosis depends upon the nature of the primary lesion which has induced the paralysis. The most unfavourable cases are those which result from the pressure of incurable tumours, from caries of the temporal bone, fractures of the bone, gunshot injuries, bulbar paralysis, and tumours of the brain. Those which result from apoplexy and embolism generally disappear in a few weeks. In syphilitic facial paralysis the prognosis is favourable, although it is not always curable. When the paralysis results from otitis interna and parotitis the prognosis depends upon the curability of these affections. Perfect recovery usually takes place after simple section of the nerve or of its branches. In rheumatic facial paralysis the prognosis mainly depends upon the character of the electrical reactions obtained.

If the electrical irritability of the paralysed nerve and muscles be normal at the end of the first week, recovery takes place in two or three weeks; and if the reaction to both currents be slightly diminished at the end of the week, recovery may be expected in the course of from four to six weeks; but if the electrical irritability be greatly diminished or completely lost at the end of the first week, the disease will last several months, and subsequent contractures will take place.

Facial paralysis of new-born infants is generally very transitory, its duration varying from a few hours to a few weeks. Duchenne has observed two cases in which the paralysis persisted in the one to 5½ and in the other to 15 years of age; but such cases are very exceptional.

§ 246. *Treatment*.—An endeavour must first be made to remove the cause of the affection. When the disease results from otitis interna and fractures of the skull, the usual surgical remedies must be adopted; and in syphilitic paralysis mercury or iodide of potassium must be prescribed. In rheumatic paralysis antiphlogistic treatment must first be adopted, consisting of the vapour bath, or a simple warm bath with subsequent warm local



fomentations. Blistering should not be used until the disease has become more or less chronic. When there is neuritis the galvanic current may be employed at an early period, the best method being the stabile application of the anode to the mastoid process of the affected side, the cathode being placed on the opposite side. After a time the cathode and anode may be alternately applied on the diseased side.

In the later stages of the affection the application of electricity forms the best direct treatment. In the slighter forms of the disease a moderately strong faradic current may be applied for two or three minutes, either daily or on alternate days, and

FIG. 37.



FIG. 37.—Motor points of face, showing the position of the electrodes in electrization of the facial nerves and muscles. The anode is placed in the mastoid fossa, and the cathode upon the part indicated in the figure.

this is generally followed by prompt improvement. The same treatment is indicated when the disease is of somewhat greater intensity, and, in addition, the galvanic current may be passed through the mastoid process in the manner already described. In the severe form the galvanic gives better results than the faradic current. It should be applied once or twice a week at first to the branches of the nerve, and to the muscles; but as soon as there are any indications of the return of motor power the application may be made more frequently. The annexed diagram (*Fig. 37*) indicates the motor points of the facial nerve.

When electricity has proved useless, little or no benefit will be obtained from the subcutaneous injection of strychnia, stimulating liniments, cold and warm douches, or any form of counter-irritation. Iodide of potassium in large doses is the only medicine which appears to exert a favourable influence in the early stages of this disease.

Various mechanical means have been used in the treatment of secondary contractures, such as traction with the fingers, and placing wooden or indiarubber balls in the cheeks. Faradisation of the healthy antagonists has also been employed as a means of exercising traction on the affected muscles.

Various operative procedures have been undertaken in incurable cases, in order to remove distortions, such as subcutaneous section of the levator palpebræ superioris, and the operation for ectropion.

### (III).—DISEASES OF THE HYPOGLOSSAL NERVE.

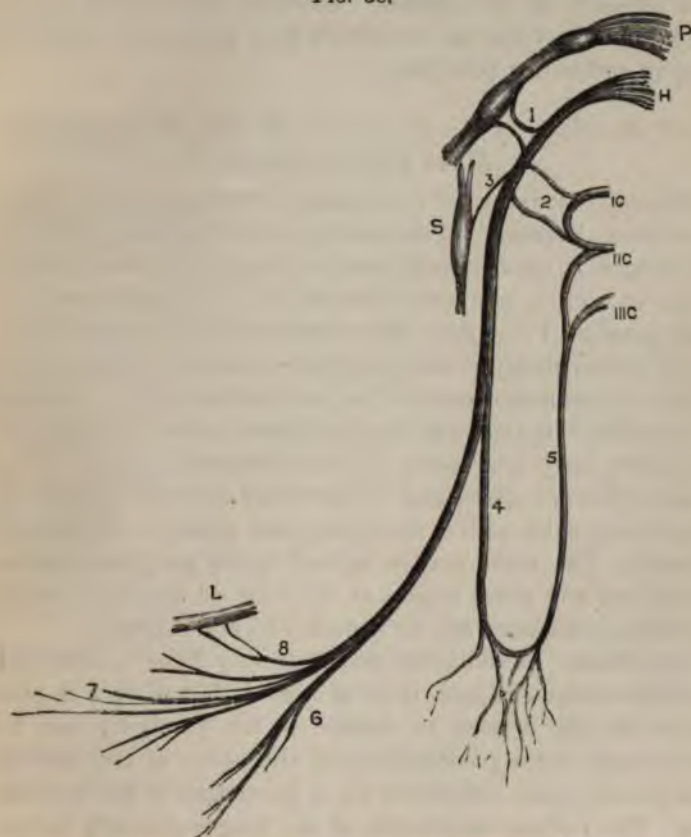
The hypoglossal is the motor nerve of the tongue, and of some of the muscles of the neck. Its course, distribution, and connections are illustrated in the subjoined diagram (*Fig. 38*).

- § 247. *Spasm of the Muscles supplied by the Hypoglossal Nerve—Lingual Spasm.*—Lingual spasm is rare as an independent affection, but it is a common symptom of hysterical convulsions, chorea, epilepsy, eclampsia, and often accompanies trismus, spasm of the facial, trigeminal neuralgia, and stuttering. It may also result from meningitis, and from lesion of the cortical motor centre of the movements of the tongue, and tremor of the tongue is a symptom of progressive muscular atrophy and bulbar paralysis.



Eulenburg quotes several cases of lingual spasm observed by various authors. In a case observed by Fleury, whenever the patient endeavoured to speak, or even formed the intention to speak, the muscles, innervated by the hypoglossal nerve, were

FIG. 38.

FIG. 38. *Diagram of the Hypoglossal Nerve, its connections and branches.*

H, Hypoglossal nerve.

P, Pneumogastric nerve.

S, Superior cervical ganglion of the sympathetic.

L, Lingual nerve.

IC, IIC, IIIC, The three upper cervical nerves.

1, Communicating branches from hypoglossal to ganglion of the trunk of the vagus.

2, Connecting filaments with the loop of first and second cervical nerves.

3, Branch to the sympathetic.

4, Descendens noni.

5, Branch from second and third cervical nerves (communicantes noni).

6, Branch to thyro-hyoid.

7, Terminal muscular branches.

8, Communicating branch to lingual branch of the fifth.

thrown into a state of clonic or tonic convulsions. In a case reported by Valleix, the tongue was fixed to the hard palate by a tonic spasm, while in a case observed by Panthel the sterno-hyoid, thyro-hyoid, and sterno-thyroid muscles were affected with clonic spasms, which could be temporarily suppressed by local pressure on the muscles. Fibrillary contractions of the lingual muscles occur in connection with progressive muscular atrophy and bulbar paralysis.

§ 248. *Paralysis of the Muscles supplied by the Hypoglossal Nerve (Glossoplegia).*

*Etiology.*—Paralysis of the muscles of the tongue is frequent in cerebral affections, but rare as the result of peripheral lesions. The tongue is usually implicated in hemiplegia from hæmorrhage, embolism, and other lesions in the hemispheres and basal ganglia. Paralysis of the tongue is also a frequent symptom of bulbar paralysis and progressive muscular atrophy; and injury of the upper part of the vertebral column, as fracture of the atlas, may implicate the hypoglossal nerve. Paralysis of this nerve may also occur in the advanced stage of tabes dorsalis (Vidal, Cuffer), and in secondary descending sclerosis, when the ganglion cells of the hypoglossal nuclei are implicated (Grasset). The nerve may be injured in its peripheral course by tumours and other lesions at the base of the brain, or by extracranial tumours, and by wounds and other injuries.

*Symptoms.*—In unilateral paralysis very little is observed when the tongue is in a state of repose; but when it is protruded the tip is seen to deviate to the paralysed side in consequence of the predominance of the action of the healthy genioglossus which directs the tip of the tongue to the opposite side. The various movements of the tongue can only be imperfectly or not at all performed on the affected side.

When the paralysis is double and complete, the tongue lies immovable on the floor of the cavity of the mouth; it is relaxed, often atrophied, with its surface wrinkled, and frequently presenting slight fibrillary contractions of its surface.

If the paralysis is incomplete, the tongue can be protruded; but complicated movements, such as raising the tip to the roof of the mouth, are impossible. Mastication is to some extent



interfered with, because the food can no longer be rolled about in the mouth and placed between the teeth; whilst deglutition is impeded, because the bolus cannot be properly collected on the dorsum of the tongue and pushed backwards into the pharynx. The consequence is that, during deglutition, food and fluids regurgitate into the mouth, and the patient is annoyed by the constant accumulation of saliva in the mouth. Articulation becomes difficult and indistinct, difficulty being first experienced in pronouncing the letters s, sch, l, e, i, and at a later period k, g, r, &c.

In bilateral paralysis with atrophy of the tongue speech becomes completely inarticulate and unintelligible; but this condition must be carefully distinguished from dumbness, aphonia, aphasia, and stuttering. Singing is difficult or impossible even in slight degrees of lingual paresis.

*Diagnosis.*—The diagnosis of lingual paralysis, as a rule, presents no difficulty. The nature of the primary lesion must be distinguished from the concomitant symptoms, as, for instance, unilateral paralysis of the tongue in association with hemiplegia indicates a cerebral origin; while bilateral paralysis in connection with paralysis of the lips and soft palate indicates a bulbar origin.

*Prognosis.*—The prognosis will depend upon the nature of the primary lesion.

*Treatment.*—The treatment must first be directed against the primary cause of the paralysis. Electricity is the most useful means of direct treatment. The galvanic or faradic current may be applied directly to the tongue or to the hypoglossal nerve in the neck, immediately above the great cornu of the hyoid bone.

## CHAPTER V.

DISEASES OF THE MIXED CRANIAL NERVES (TRIGEMINUS,  
PNEUMOGASTRIC, SPINAL ACCESSORY).

## (I.)—DISEASES OF THE TRIGEMINAL NERVE.

THE sensory portion of the fifth nerve subdivides into three main branches, named respectively the first, or ophthalmic; the second, or superior maxillary; and the third, or inferior maxillary, the last being joined by the smaller or motor root of the nerve. The terminal distribution of the different subdivisions is represented in *Fig. 35*; while the course and branches of the divisions of the nerve are shown diagrammatically in *Figs. 36, 37, and 38*.

§ 249. *Functions*.—The sensory fibres of the nerve confer sensibility on nearly the whole of the head. The regions of the head not supplied by the fifth are: portions of the pharynx, palate, root of the tongue, the Eustachian tube, the tympanic cavity, and a part of the external auditory meatus and external ear which receive branches from the vagus and glosso-pharyngeal nerves; whilst a portion of the back of the head and the region of the parotid are supplied by the cervical spinal nerves.

*The lingual branch* of the fifth has been considered to be the special nerve of taste for the anterior two-thirds of the tongue, but it has already been pointed out that the fibres presiding over this function join it from the chorda tympani.

The motor part of the nerve supplies the temporal, both pterygoids, masseter, mylo-hyoid, and the anterior belly of the digastric.

According to some it supplies fibres to the dilator iridis; whilst the tensor tympani and tensor palati receive motor filaments from the otic ganglion.



*Vaso-motor fibres*, probably of sympathetic origin, run in the trigeminus for the conjunctiva and iris.

Secretory fibres are also contained in it for the lachrymal, parotid, and submaxillary glands.

*Trophic fibres* destined for the eyeball appear to be contained in it, inasmuch as section of the fifth in the skull causes inflammation and degeneration of that organ. It is also said to contain trophic fibres for the cavity of the mouth, because section of the nerve occasions ulceration of the oral mucous membrane.

FIG 39.

FIG. 39. *Nerves of the Face and Scalp* (from Hirschfeld and Leveillé).

- |   |   |
|---|---|
| 1, Attrahens aurem muscle.  | 17, Facial nerve (7th).                             |
| 2, Anterior belly of occipito-frontalis.                          | 18, Nasal nerve (5th).                              |
| 3, Auriculo-temporal nerve.                                       | 19, Cervico-facial division of 7th.                 |
| 4, Temporal branches of facial nerve (7th).                       | 20, Infra-orbital nerve (5th).                      |
| 5, Attollens aurem muscle.  | 21, Branches to digastric and stylo-hyoid.          |
| 6, Supra-trochlear nerve (5th).                                   | 22, Temporo-facial division of 7th.                 |
| 7, Posterior belly of occipito-frontalis.                         | 23, Great auricular nerve.                          |
| 8, Supra-orbital nerve (5th).                                     | 24, Buccal branches of facial nerve.                |
| 9, Retrahens aurem muscle.  | 25, Trapezius.                                      |
| 10, Temporal branch of orbital nerve (5th).                       | 26, Buccinator nerve (5th).                         |
| 11, Small occipital nerve.  | 27, Splenius capitis.                               |
| 12, Malar branches of facial nerve.                               | 28, Masseter.                                       |
| 13, Posterior auricular nerve (7th).                              | 29, Sterno-mastoideus.                              |
| 14, Malar branch of orbital nerve (5th) (ramus subcutaneus malæ). | 30, Supra-maxillary branches of facial nerve (7th). |
| 15, Great occipital nerve.  | 31, Superficial cervical nerve.                     |
| 16, Infra-orbital branches of facial nerve (7th).                 | 32, Mental nerve (5th).                             |
|   | 33, Platysma.                                       |
|   | 34, Infra-maxillary branches of facial nerve (7th). |

§ 250. *Anæsthesia of the Trigemini.*—Anæsthesia of the fifth, or of one or more of its branches, may be caused by pathological changes in the nerve or ganglion, intracranial tumours, diseases of the bones, vessels, and membranes of the brain, fractures of the skull, and wounds of the head. It is scarcely necessary to point out that the lesion may be situated in any part in the course of the nerve from its deep origin in the nuclei of the floor of the fourth ventricle to its final distribution.

§ 251. *Symptoms.*—When all the branches of the nerve are affected, one side of the face, part of the ear, the skin of the temple and fore part of the head, the conjunctiva, cornea, nasal and oral mucous membranes, the tongue, gums, and part of the pharynx are all rendered more or less completely insensitive on the affected side. When the patient puts a cup to his lips it gives him the impression of being broken, as he can only feel with one half of the lip; violent eccentric pains may also be felt (*anæsthesia dolorosa*). The movements of mastication are affected when the motor root is implicated; and although the muscles supplied by the seventh are not paralysed, yet the facial movements on the anæsthetic side are often slow and imperfect. When the anæsthesia is of peripheral origin reflex actions are abolished.

The skin of the face is cold, and often of a bluish colour. The gums of the affected side are spongy, the mucous membrane of the mouth and nose of that side may ulcerate and bleed, and neuro-paralytic ophthalmia may occur. Irritation of the nasal mucous membrane by ammonia or snuff does not excite a reflex action; and the sense of smell on the same side is also diminished, probably owing to dryness of the Schneiderian membrane. The sense of hearing is unaffected provided the tensor tympani be not paralysed, but the sense of taste is lost in the anterior two-thirds of the anæsthetic side of the tongue. The extent of the anæsthesia will of course vary according to the seat of the disease, and it may sometimes be limited to the area of distribution of a single branch of the nerve.

§ 252. *Diagnosis.*—The chief difficulty in diagnosis is to discover the locality of the primary lesion, and more especially



to determine whether the affection be of peripheral or central origin. The following rules laid down by Romberg may be of use in determining this question :—

(a) The more the anæsthesia is confined to single filaments of the nerve, the more peripheral will be the lesion.

(b) If the anæsthesia is found in a portion of the face, and in the oral cavity, the disease may be inferred to affect the sensory fibres of the fifth before it divides; or, in other words, a main division must be affected before or after its passage through the cranium.

(c) When the distribution is diffused over the entire area of distribution of the fifth, and there are concomitant trophic changes, the Gasserian ganglion, or the nerve in its immediate vicinity, is the seat of the disease.

(d) If the anæsthesia of the fifth nerve be complicated with disturbed function of adjoining cerebral nerves, as the third or sixth, or white atrophy of the optic nerves, then the disease may be inferred to be situated at the base of the brain.

(e) The diagnosis of true central anæsthesia of the fifth is established by the coincident affections of other nerves of the face, trunk, and extremities.

§ 253. *The treatment* of anæsthesia of the fifth nerve must be conducted on the same principles as that of other forms of anæsthesia, *i.e.*, by endeavouring to remove the cause, and by direct treatment. In applying electricity to the face the usual precautions must be taken not to use too strong a current.

§ 254. *Neuralgia of the Fifth Nerve (Trigeminal Neuralgia).* Trigeminal neuralgia consists of paroxysms of neuralgic pain in the region of distribution of the fifth nerve or of some of its branches.

§ 255. *Etiology.*—The general causes of neuralgia of the fifth, being the same as the general causes of all forms of neuralgia, they need not be recapitulated. The female sex appears to be more susceptible to trigeminal neuralgia than to any other superficial form of the disease, except mammary neuralgia. It also occurs more frequently than any other form of neuralgia in epileptic

families. Anæmia, arterial degeneration, wounds, cicatrices, and diseases of neighbouring tissues, exercise the same influence in neuralgia of the fifth as in the neuralgias of other nerve territories. The passage of this nerve through bony canals, however, renders it very liable to be implicated in various affections of the bones and periosteum. Aneurism of the internal carotid has been known to cause intractable neuralgia by pressure on the Gasserian ganglion. The important special causes of facial neuralgia are peripheral irritations, as carious teeth, retarded appearance and false development of the wisdom teeth, disease of the nasal and frontal sinuses, and over-exertion of the eyes the last of which was regarded by Dr. Anstie as the most fruitful cause of trigeminal neuralgia.

Neuralgia of the fifth may also be caused by injury of remote nerves. Dr. Anstie observed one case in which injury to the occipital and another to the ulnar nerve seemed to have caused neuralgia of the fifth. Intestinal worms, and various genital irritations in both sexes, excessive mental strain, and especially severe emotional disturbances, may be mentioned as amongst the most important of the indirect exciting causes of trigeminal neuralgia.

§ 256. *Symptoms*.—The attack may come on suddenly, but the patients often complain for hours or days, before the actual paroxysm, of feelings of pressure and tension, stiffness, itching, and formication on the side of the face threatened with the attack; while, in other instances, the attack is preceded by a general feeling of malaise, shivering, and flying pains about the teeth. During the attack continuous pain is felt throughout the whole of the affected region, being now and then interrupted by lightning-like darts of pain. In the more severe cases these lightning-like darts recur so frequently as to give rise to more or less constant pain, intolerable in its severity, and defying all description. The paroxysms recur at varying intervals, according to the nature of the case, the state of the general health, the season of the year, and various other circumstances. In cases of great severity the slightest external injury, as a draught of cold air, washing or shaving the face, sneezing, or any slight emotional disturbance, may induce a paroxysm of the disease.



*Painful points* are observed during the attacks, and sometimes even in the periods of intermission, corresponding generally to the localities where the nerve becomes more superficial, either in issuing from a bony canal, or in penetrating fasciæ.

*The concomitant symptoms* of trigeminal neuralgia are more numerous and varied than those of any other form of the disease. Irradiation of the pain to other nerve territories usually accompanies a severe paroxysm.

When one branch of the fifth is affected, the pain extends during the paroxysm to the other branches of the same nerve, or to the occipital nerves, and it may extend in severe cases to the neck, shoulders, any part of the area of distribution of the intercostal nerves, especially the mammary gland. In some cases it may even extend to the extremities.

*Either hyperæsthesia or anæsthesia* of the skin of the affected part is almost always present, the rule being, although it is not without exceptions, that hyperæsthesia is observed in recent, and anæsthesia in chronic cases.

*Disturbances of the organs of special sense* have in some rare instances been observed. Photopsia, amblyopia, and even amaurosis, along with certain disorders in the sense of hearing, have been described by some authors as the result of facial neuralgia. Affections of the gustatory and olfactory nerves also, are occasionally mentioned.

*Spasm of various* muscles is the most usual concomitant motor phenomenon, and the muscles supplied by the facial nerve suffer most. The contraction of the facial muscles sometimes appears in the form of blepharospasm, sometimes as a contraction of the muscles at the angle of the mouth, or as true convulsive tic. Both tonic and clonic spasms of the muscles of mastication have been observed, but these cases are rare, and paralysis of the muscles is still rarer. Spasmodic movements of the tongue have been observed in some severe paroxysms. These spasms may extend to the muscles of the whole body, and in hysterical subjects a neuralgic paroxysm may be the starting point of general convulsions. Paralysis of some of the ocular muscles has been described in connection with facial neuralgia, but it would probably be found that such

cases, if carefully investigated, had been caused by the pressure of an intracranial tumour on the cavernous sinus.

*Vaso-motor* disturbances are of common occurrence. In the first stage of the attack the affected side of the face presents an abnormal pallor; but at a subsequent stage the pallor is replaced by an intense red, the skin presenting a polished appearance with slight cedematous swelling. The redness extends to the mucous membranes supplied by the fifth, and the conjunctiva is specially affected and presents a high degree of hyperæmia. Visible perspiration and strong pulsation of the carotid, facial, and temporal arteries of the affected side of the face may also be observed during the attack.

*Secretory disturbances* are, as might have been anticipated, also common, and increase of the lachrymal secretion is by far the most frequent of these. Both the lachrymal and orbital nerves are known to contain secretory fibres for the lachrymal gland; and irritation of the sensory branches of the first and second divisions of the fifth increases the flow of tears by reflex action. Augmented salivary secretion is sometimes present, and occasionally there is an increased secretion from the nasal mucous membrane of the affected side.

*Trophic disturbances* are of various forms in connection with trigeminal neuralgia. The most usual of these are swelling of the face, changes in the colour and texture of the hair, herpetic eruptions, erysipelas, subacute inflammation of the periosteum and of the fibrous membranes in the neighbourhood of the painful points, neuroparalytic ophthalmia, and, according to Anstie, iritis and glaucoma.

*The psychical disturbances* present are mental irritability and despondency, and occasionally patients, in order to escape from the severe suffering, have committed suicide. The great suffering and sleeplessness in some cases lead to impairment of nutrition and exhaustion.

#### VARIETIES OF TRIGEMINAL NEURALGIA.

§ 257. *Neuralgia of the First Branch of the Fifth (Ophthalmic Neuralgia).*—The several branches of the ophthalmic division of the fifth nerve may either all be affected or the neuralgia may be limited to some particular branch. The



painful points in connection with the ophthalmic division of the nerve are :—

(1) The *supra-orbital* at the supra-orbital foramen, or a little higher, in the course of the frontal nerve ; (2) the *palpebral*, in the upper eyelid ; (3) the *nasal*, at the point of emergence of the long nasal branch, at the junction of the nasal bone with the cartilage ; (4) the *ocular*, a somewhat indefinite focus within the globe of the eye, when the ciliary nerves are affected ; (5) the *trochlear*, at the inner angle of the orbit.

Supra-orbital neuralgia is the form which generally results from exposure to cold ; and it is, also, almost the exclusive seat of malarial neuralgia. The characteristic features of this form of the affection are pain in the forehead extending downwards to the upper eyelid and root of the nose, hyperæmia of the conjunctiva, lachrymation, and in almost all instances there is a well-marked painful spot at the supra-orbital foramen.

FIG. 40.

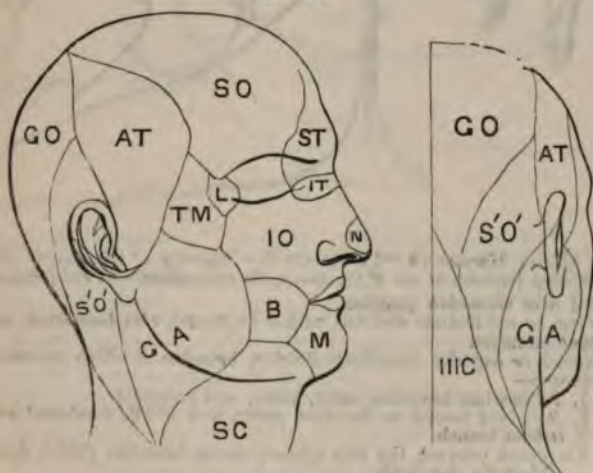


FIG. 40 (after Flower).—*Sensory Nerves of the Head and Face.*

First division of the fifth :

SO, Supra orbital.  
ST, Supra trochlear.  
IT, Infra trochlear.  
L, Lachrymal.  
N, Nasal.

Second division of the fifth :

IO, Infra orbital.  
TM, Temporo malar.

Third division of the fifth :

B, Buccal.  
M, Mental.  
AT, Auriculo temporal.

Branches of the cervical plexus :

GO, Great occipital.  
S'O', Small occipital.  
GA, Great auricular.  
SC, Superficial cervical.  
IIIC, Third cervical.

§ 258. *Neuralgia of the Second Division of the Fifth Nerve (Supra-maxillary Neuralgia).*—When all the branches of the nerve are affected the pain is situated in the cheek, eyelid,

FIG. 41.

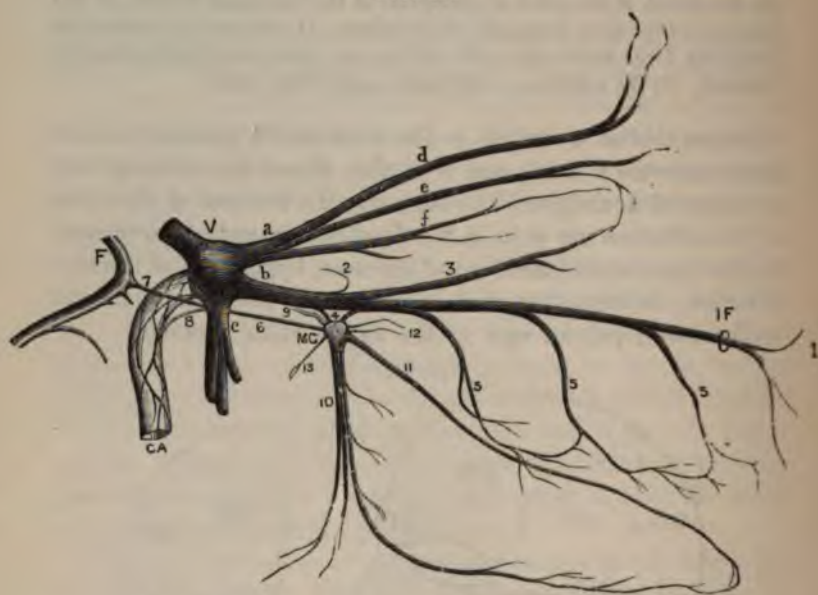


FIG. 41 (From Hermann's "Physiology").—Diagram of the Second (Superior Maxillary) Division of the Fifth Nerve, its connections and chief branches.

V, Placed over Gasserian ganglion.

a, First or ophthalmic division, with *d* its frontal, *e* its lachrymal, and *f* its nasal branches.

b, Second or superior maxillary division, branches of which are marked as follows:—

1, Its terminal branches, nasal, labial, and palpebral.

2, Recurrent branch to the dura mater, and middle meningeal artery.

3, Orbital branch.

4 is placed between the two *spheno-palatine* branches (which descend to Meckel's ganglion).

5, Dental branches.

MG, Meckel's ganglion.

6, The Vidian nerve (constituting the motor and sympathetic root of Meckel's ganglion).

7, The *great superficial petrosal* nerve, from the geniculate ganglion of the facial nerve, joining the Vidian.

8, The *sympathetic* branch from the plexus on the carotid artery, joining the *great superficial petrosal*, and forming with it the Vidian nerve.

9, Ascending branches of Meckel's ganglion.

10, Descending palatine branches.

11, Naso-palatine branch.

12, Upper nasal branches.

13, Pharyngeal branch.

F, Facial nerve. CA, Carotid artery. IF, Infra-orbital foramen.



lateral portion of the nose and upper lip (infra-orbital nerve), in the zygomatic arch and anterior temporal region (orbital

FIG. 42.

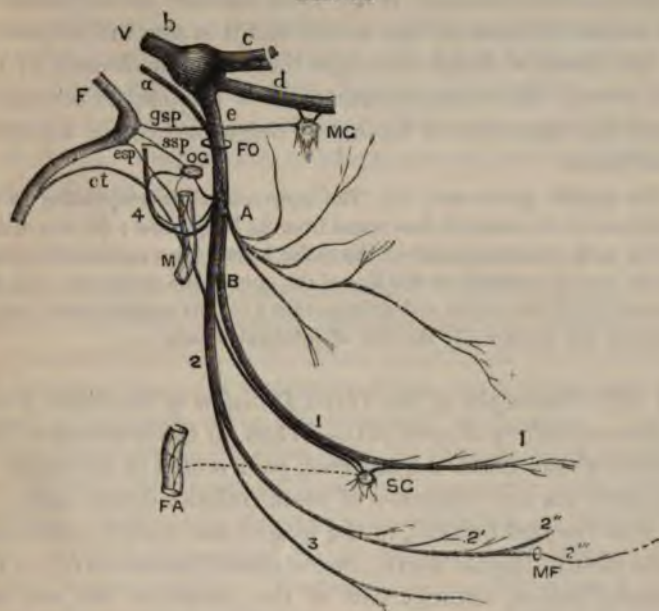


FIG. 42 (From Hermann's "Physiology").—*Diagram of the Third (Inferior Maxillary) Division of the Fifth Nerve, its connections and chief branches.*

V, Fifth nerve. *b*, Its largest sensory root, with the Gasserian ganglion.

*a*, Its smaller motor root joining *e*, the third division of the Gasserian ganglion, to form the inferior maxillary nerve.

A, Anterior division of inferior maxillary nerve (mainly motor) supplying branches to the muscles of mastication, and a terminal buccal branch to the mucous membrane of the mouth.

B, Posterior division (mainly sensory); its branches are marked—

1, Lingual nerve; 1', Branches to the tongue.

2, Inferior dental nerve; 2', Its twigs to the teeth; 2'', Incisor branch; 2''', Mental branch.

3, Mylo-hyoid branch to digastric and mylo-hyoid.

4, Auriculo-temporal nerve.

F, Facial nerve. *ct*, Its chorda tympani branch, joining the lingual, and running to the submaxillary ganglion SG, of which it forms the motor root.

OG, Otic ganglion:

*esp*, Small superficial petrosal nerve, connecting otic ganglion and facial nerve.

M, Middle meningeal artery, from the plexus upon which sympathetic filaments pass to the otic ganglion;

*esp*, External superficial petrosal nerve, connecting the plexus on the middle meningeal artery with the facial nerve;

*gsp*, Great superficial petrosal nerve, connecting the facial with Meckel's ganglion.

FA, Facial artery, from the plexus upon which sympathetic filaments pass to the submaxillary ganglion.

FO, Foramen ovale. MF, Mental foramen.

SG, Submaxillary ganglion.

nerve), in the upper row of teeth (dental branches), in the nasal cavities and gums (naso-palatine and posterior palatine nerves).

Infra-orbital neuralgia is the most common variety affecting the second division of the nerve, and it is also the severest of all the forms of facial neuralgia limited to one branch of the fifth nerve. The characteristic pain is localised in the cheek, upper lip, upper row of teeth, and neighbourhood of the zygomatic arch.

The painful points are : (1) The *infra-orbital*, corresponding to the emergence of the nerve of that name from its bony canal ; (2) the *malar*, on the most prominent part of the malar bone ; (3) a vague and indeterminate focus somewhere on the line of the gum of the upper jaw ; (4) the *superior labial*, also vague and unimportant ; (5) the *palatine point*, rarely observed, but occasionally the seat of intolerable pain.

§ 259. *Neuralgia of the Third Division of the Fifth Nerve (Infra-maxillary Neuralgia).*—When all the branches of this division of the nerve are affected, pain occurs in the region of the lower jaw and lower row of teeth (inferior dental nerve), in the chin (mental branch), in the tongue and mucous membrane of the mouth (lingual nerve), in the cheek (buccal nerve), in the temporal region, anterior part of the auricle of the ear, and external auditory meatus (auriculo-temporal nerve).

The painful points are : (1) *The temporal*, a point on the auriculo-temporal branch, a little in front of the ear ; (2) *the inferior dental point*, opposite the point of emergence of the nerve of that name ; (3) *the lingual point* on the side of the tongue, but it is rarely met with ; (4) *the inferior labial point*, also rarely observed. Besides the painful points which are in relation with distinct branches of the trigeminus, there is a point situated a little above the parietal eminence which is more commonly affected in facial neuralgia than any other point. *The parietal point* corresponds to the inosculation of various branches of the nerve.

The cases of facial neuralgia which have been described by Trousseau under the name of "Epileptiform Neuralgia," and which Benedict regarded as the only true examples of "*tic douloureux*," are extremely severe and obstinate. Pains of the most violent and lightning-like nature succeed each other with the greatest rapidity for a few seconds or minutes, and then suddenly vanish. But these short attacks may recur and follow



each other in quick succession for a period of hours, days, or even weeks; then follows a respite, and the paroxysm disappears for days, weeks, or even years, although relapses are sure to take place after a longer or shorter time. This form of neuralgia is of centric origin; it occurs in families, manifesting a proclivity to psychoses, and is frequently accompanied by epilepsy; or it is associated with a high degree of excitability and with strongly-marked mental activity. The prognosis of this form of neuralgia is unfavourable, although not so grave as Trousseau believed it to be.

§ 260. *Course, Duration, and Terminations.*—The slighter forms of facial neuralgia may last a few hours only, but the severer forms of *tic douloureux* continue to affect the patient for the remainder of his life. The great majority of cases terminate in recovery, the most favourable being those which arise from malaria, exposure to cold, rheumatism, or anæmia. The unfavourable cases, on the other hand, are those in which there is a well marked hereditary predisposition, especially if the patient, or some members of his family, be epileptics, or those which come on at a late period of life, associated with arterial degeneration of arteries. The disease is incurable when it occurs from severe organic affections, and also in some of the cases which Trousseau has called epileptiform neuralgia.

§ 261. *Diagnosis.*—Facial neuralgia may be confounded with toothache, but in the latter the pain is continuous, and one or more of the teeth will be found carious, and sensitive to cold and mechanical shock. It may also be mistaken for inflammation of the facial bones, and of the membranes lining the antrum and frontal sinuses, but in these cases there must be some degree of tumefaction and general tenderness to pressure, while the situation and kind of pain do not correspond to that of any of the branches of the fifth nerve. Hysterical clonus is distinguished from neuralgia by being limited to one spot from which the pain does not radiate, and by the presence of other hysterical symptoms.

In anæmic or dyspeptic headache, the pain has no fixed position, it is deep-seated, dull, tensive, and does not increase

in paroxysms; while in migraine the pain is deeply seated in the skull, pulsating, accompanied by vomiting and much mental irritability, and does not correspond with any special branch of nerve.

It is important to distinguish between neuralgia of peripheral and of central origin. Limitation of the pain to a definite branch of the nerve, the presence of some manifest peripheric cause, the possibility of cutting short the attack by remedies applied to the periphery, and the presence of painful points in the intervals between the paroxysms may be taken as signs that the disease is of peripheral origin. Lancinating pains, localisation in bones, widely distributed reflex contractions, general hyperæsthesia, and well marked mental irritability, disorders of other cerebral nerves, and absence of painful points in the intervals between the attacks, may be taken as indications of the central origin of the disease.

§ 262. *Treatment.*—The treatment of facial neuralgia must be conducted on the broad principles already described, while treating of the subject of general neuralgia; and it is only necessary at present to mention a few special points with respect to the treatment of individual cases. In cases of supra-orbital neuralgia of malarial origin, large doses of quinine constitute the most effectual remedy, and often do good in other cases. One large dose of a scruple may be administered, or four grains may be given every four hours until the pain is relieved, or until headache and noises in the ears render it necessary to desist.

If the neuralgia be caused by osteitis, periostitis, or neuritis from any other cause, an energetic antiphlogistic treatment must be adopted; and in rheumatic cases diaphoresis and the internal administration of colchicum and opium, or salicylate of soda should be adopted. Every source of peripheral irritation should be removed. Amongst the most usual of these are carious teeth, foreign bodies pressing and injuring nerves, cicatrices, tumours, and neuromata, and the removal of these often leads to speedy recovery. Great attention should be paid to the general health, more especially if anæmia or hereditary predisposition be present.



*Electricity.*—The constant current has been found very successful in the treatment of obstinate neuralgias. In peripheral neuralgia of isolated superficial nerves the direction method may be employed, and a descending *stabile* current passed through the painful nerve. When the polar method is used, the anode is to be placed on the specially painful points and held stationary there, whilst the cathode rests on the back of the neck, or on any other indifferent part of the body. When the deeper-seated nerves, as the infra-orbital and lingual, are affected, the anode may be placed on the neck or behind the ear, and the cathode over the point of emergence of the affected nerve. In order to reach the main divisions of the nerve at the base of the cranium, and after their emergence through the foramina of the sphenoid bone, the current may be conducted transversely through the base of the skull at the appropriate spots, the anode being placed on the painful side. Benedict recommends that in severe cases galvanic currents should be passed longitudinally and transversely through the skull, and along the sympathetic nerve.

*Faradisation* occasionally answers better than the constant current. The moist poles should be employed and applied to the painful points and the nerve trunks. Narcotics, and more especially subcutaneous injections of morphia, are useful in this as in all other forms of neuralgia. It may be advantageous to change sometimes from morphia to stramonium, hyoscyamus, or belladonna, and each must be given in increasing doses.

Trousseau advised the treatment of epileptiform neuralgia by large doses of opium or morphia. In some cases he gave as much as a drachm of morphia, or two or three of opium in the course of the day; a treatment which is occasionally successful, but must be used with caution, and only resorted to when all other methods have failed. Ointments, containing opium, veratria, aconitia, or equal parts of chloral hydrate and camphor, may be rubbed into the skin over the painful nerve, and chloroform, both as an external application and by inhalation, has been found useful.

Quinine, arsenic, zinc, nitrate of silver, chloride of gold and sodium, strychnia, carbonate of iron, and iodide of potassium are the most useful internal remedies; and of these arsenic

is by far the most generally useful, except in neuralgia of malarial or syphilitic origin, in which case quinine or iodide of potassium must be given according to the nature of the case. *Gelsemium sempervirens*, best given in the form of tincture, and croton chloral hydrate, either in one large dose of a scruple or four grains every four hours, have also been found useful; but in my experience they are of no use in obstinate cases. When there is anæmia, carbonate of iron may be given along with cod-liver oil.

Various counter-irritants have been employed, but they do not appear to give much relief, and as they cause considerable pain they should not be needlessly employed.

Patients often experience great relief from warm fomentations and poultices; but can rarely bear the application of cold. In severe cases I have seen the constant application of an ice bag of temporary use. Continuous pressure may be tried on the nerve at the painful points.

Sea-water bathing, thermal vapour baths, and "cold water cures" are often followed by favourable results. In intractable cases, when all other methods have failed, recourse must be had to neurotomy or neurectomy; but, although in many cases immediate relief is afforded, the pains return in about three months, and the operation has to be repeated.

#### § 263. *Trophic Affections in the Territory of the Trigemini.*

Herpes zoster is frequently found in the area of distribution of single branches of the trigeminus. The eruption is generally unilateral, although in some cases it has been observed on both sides of the face. In the latter cases the disease was distributed over all the branches of the trigeminus. Neuralgia may or may not be associated with herpes. Herpes in the region of the frontal nerve is often a very severe affection, being ushered in by hemicrania, or general headache, or frontal neuralgia. When descending branches of the nerve are affected, the eruption extends over the upper eyelid and the side of the nose, and the conjunctiva becomes implicated. In severe cases the eyeball becomes affected, giving rise to inflammation, not only of the conjunctiva, but of the cornea, and even general inflammation of the eyeball may result, the most prominent feature being



iritis. That these inflammatory conditions depend upon irritation of the trigeminus, and of the Gasserian ganglion, has been proved by the post-mortem examinations of Wyss and Kaposi, who found evidence of neuritis and hæmorrhage in the ganglion. Gerhardt thinks that the herpes labialis, which is so frequently present in acute diseases, such as pneumonia, is due to irritation of single branches of the trigeminus, caused by sudden dilatation of arteries in the narrow bony canals through which the branches of the nerve pass.

§ 264. *Simple Glaucoma*.—Donders in 1863 directed attention to the fact that simple glaucoma appeared to be due to irritation of the secretory fibres which are conveyed in the trigeminus. It is maintained by Wagner, on the other hand, that the intraocular increase of pressure, on which glaucoma depends, is caused by irritation of the cervical sympathetic, in which the vaso-motor nerves are conveyed. He thinks, however, that increase of the intraocular pressure may be caused in a reflex manner by irritation of the sensory trigeminus branches. The experiments of Hippel and Grünhagen show that irritation of the trigeminus nucleus in the medulla oblongata is followed by a considerable and enduring increase of the intraocular pressure; and the same result is produced by the introduction of nicotine into the eye through peripheral trigeminal irritation. According to these experiments, glaucoma may be produced either by central or peripheral irritation of the trigeminus. The increase of the intraocular pressure is caused by increased secretion of the aqueous humour. The results of this high tension are that the iris and lens are pushed forwards, and the internal membranes are stretched. The increase of pressure in the globe of the eye frequently observed in different forms of trigeminal neuralgia, with which a certain amount of deficiency of accommodation is often associated; the previous occurrence of neuralgia of the external facial branches of the trigeminus, of ciliary neuralgia and hemi-crania in simple as in inflammatory glaucoma, the return of the increased intraocular pressure with every attack of neuralgia, are decidedly in favour of the dependence of glaucoma upon irritation of the trigeminus.

§ 265. *Neuro-paralytic Ophthalmia*.—It was first shown by Majendie that intracranial destruction of the trigeminus in rabbits, especially below the Gasserian ganglion, was followed by a severe nutritive disturbance of the eye on the affected side.

The affection begins with strong congestion of the conjunctival vessels, followed by profuse secretion of mucus or pus, insensibility and opacity of the cornea, redness and pseudo-membranous exudation of the iris. In from five to ten days ulceration and perforation of the cornea occurs, which is followed by loss of the humours, collapse and complete destruction of the eye.

Cases of this kind have been observed in man in connection with complete or incomplete anæsthesia of the trigeminus.

Three suppositions in explanation of these phenomena are possible :—

(1) That the lesion of the eye results from the fact that the presence of anæsthesia does not enable it to protect itself against external injury. (2) That it is due to paralysis of vaso-motor fibres contained in the trigeminus. (3) That it is due to injury of trophic fibres, and is analogous to the cutaneous trophic affections already described. The first supposition was supported by Snellen and Büttner, but it is contradicted by the facts that, in complete anæsthesia of the eye, this affection may fail to appear; and, conversely, the nutritive affection may appear in neuralgia or partial destruction of the trigeminus when the conjunctival sensibility is completely maintained. In paralytic lagophthalmos from paralysis of the facial, when the eye is in the highest degree unprotected from external sources of irritation, the severe affection observed in trigeminal injury altogether fails.

The vaso-motor theory is rendered improbable by the facts that extirpation of the superior cervical ganglion not only does not cause ophthalmia, but even diminishes the consequences which follow from subsequent destruction of the trigeminus; or improves the symptoms already caused by a previous injury to the fifth nerve (Sinitzin).

The third supposition, attributing the ophthalmia to an irritation of trophic fibres which probably descend from the Gasserian ganglion, is by far the most probable.

The most frequent causes of this affection in man are intracranial tumours and tubercular meningitis, which attack the Gasserian ganglion or invade the origin of the fifth nerve.

#### § 266. *Spasm in the Region of Distribution of the Trigeminal Nerve. Masticatory Spasm. Trismus.*

In masticatory spasm the affection is limited to the muscles



supplied by the motor division of the fifth nerve. The spasm may be either *tonic* or *clonic*, and it is as a rule bilateral.

*In the tonic variety* the lower jaw is approximated to the upper, and in the severe form of the affection the teeth are so powerfully clenched that they cannot be separated from one another by force, constituting the condition termed lock-jaw or *trismus*. The muscles of the jaw are tense, rigid, often painful, and mastication is impossible.

*In the clonic form* the lower jaw is moved either in a vertical or horizontal direction; the former giving rise to chattering of the teeth, as in the cold stage of ague, and the latter to grinding of the teeth accompanied by munching movements. The depressors of the jaw are sometimes subject to spasm, which is usually of the tonic variety. Associated symptoms, depending on the primary lesion, are also usually present. The most common of these are trigeminal neuralgia, toothache, symptoms of periostitis of the lower jaw, and various cerebral symptoms. The sequelæ to masticatory spasm are such as biting of the tongue and lips, ulcerations and inflammation of the gums and mucous membrane of the mouth, and inanition consequent upon deficient supply of nourishment. The teeth are usually much worn in the clonic variety by the constant grinding against each other, and they may even be broken by the violence of the spasm.

*Etiology.*—Bilateral masticatory spasm is often a concomitant symptom of general spasms, such as tetanus, epilepsy, eclampsia, hysteria, and chorea. At other times it occurs as a separate symptom. The affection may then be caused by diseased conditions of the nerves themselves, such as neuritis, softening, tumours; or it may be a symptom of basal meningitis, apoplexy, intracranial tumours, and other central affections.

Masticatory spasm also frequently results from reflex irritation. The peripheral irritation may either be in the region of distribution of one of the sensory branches of the fifth, as the variety caused by carious teeth; but frequently the irritation is at a distance, as in the extremities, or in the intestines from the presence of worms.

*Diagnosis.*—The tonic form might possibly be mistaken for ankylosis of the lower jaw, but the two affections can be

readily distinguished by a careful examination, and in cases presenting unusual difficulties chloroform will aid the diagnosis.

*Prognosis.*—The prognosis is generally favourable in the forms which arise from exposure to cold, and from reflex irritation; but it is unfavourable in diseases of the central nervous system, in severe general convulsions, and especially in tetanus.

*Treatment.*—The treatment must be directed to the removal of the cause of the affection. In rheumatic cases, or those which arise from exposure to cold, active diaphoresis, opium, and iodide of potassium may be tried. In reflex masticatory spasm the peripheral source of irritation must be discovered and removed.

Electricity is the most valuable agent for direct treatment, and the usual methods of applying the galvanic or faradic currents must be employed.

Subcutaneous injection of morphia is very useful; and in obstinate cases nervine tonics may be given, such as zinc, arsenic, or iodide of potassium. The diet must be carefully regulated, and it is requisite to give nourishment in a fluid form, either by means of a tube introduced through a space in the teeth or through the nose, or by the use of nutritive enemata.

In chronic cases an attempt has been made gradually to separate the jaws by mechanical means, as by the daily introduction of progressively thicker wedges of wood between the teeth. In a case recently under my care, which resisted ordinary treatment, chloroform was administered, and the jaws forcibly separated by means of a gag. The spasm did not subsequently recur.

§ 267. *Paralysis in the Region of Distribution of the Fifth Nerve. Masticatory Paralysis.*

*Etiology.*—Paralysis of the motor branch of the trigeminus is rare from lesion in its extracranial course, because the nerve is so deeply situated and so well protected from injuries and exposure to cold. The most frequent causes of masticatory paralysis are intracranial lesions, which compress the motor branch of the nerve at the base of the skull, such as periostitis, exostoses, caries, extravasations, and tumours of all sorts.



Paralysis of this nerve is also caused by lesions of the pons and upper part of the medulla oblongata affecting the motor root of the trigeminus.

*Symptoms.*—Where the paralysis is unilateral there is difficulty or impossibility of masticating food on that side, and the lateral movements towards the sound side are rendered impossible by paralysis of the pterygoids. During mastication it may be both seen and felt that the muscles on the affected side do not enter into contraction; while those of the opposite side may be seen to swell up, and feel rigid at each contraction.

When the affection is bilateral the patient suffers great fatigue during mastication, and he is forced to eat only fluid and pulpy nourishment; and when the paralysis is complete the lower jaw falls down with its own weight. At times the jaw may be fixed by secondary contracture of the paralysed muscles. The muscles may undergo the reaction of degeneration to the galvanic and faradic currents, and the paralysis is frequently associated with atrophy of the muscles.

The tensor veli palati and the tensor tympani are also involved in the paralysis. Unilateral paralysis of the tensor palati gives rise to elevation of the palate of the paralysed side owing to the action of the levator palati, but this condition has not hitherto been observed along with masticatory paralysis. Little is known of paralysis of the tensor tympani, although Romberg mentions the case of a patient who was rendered dull of hearing on the paralysed side; and Benedict mentions the case of a patient who suffered from noises in the ear of the affected side. In such cases there is diminution of hearing for deep tones, which, according to Lucæ, become better appreciated when the masticatory muscles are contracted, owing to the tensor tympani participating in the action. Masticatory paralysis is rare as a separate affection. It is usually associated with anæsthesia of the third division, or of one or more of the other divisions of the trigeminus, and with paralysis of the third and other nerves.

The diagnosis does not present any difficulty; the prognosis varies according to the cause; and the treatment comprises the ordinary means, such as electrification and other remedies.

## (II).—DISEASES OF THE VAGUS, PNEUMOGASTRIC OR TENTH CRANIAL NERVE.

The pneumogastric has the longest course of any of the cranial nerves, extending through the neck and cavity of the chest to the upper part of the abdomen. The nerve emerges from the medulla oblongata, between its lateral column and the restiform body; its roots, twelve or fifteen in number, lying beneath and in a line with the roots of the glosso-pharyngeal nerve.

(1) *Diseases of the Pharyngeal and Œsophageal Plexuses.*

The pharyngeal plexus is composed not only of branches of the vagus, but also of the glosso-pharyngeal and the sympathetic, and the relative function of each of these in the innervation of the soft palate has not yet been determined. The levator palati and azygos uvulæ are supplied from the seventh, but it will be more convenient to group together all the different forms of paralysis of the soft palate, no matter from what source the muscles derive their nerve supply. The following table gives the muscles which move the soft palate, with their origins and insertions.

<i>Name of Muscle.</i>	<i>Origin.</i>	<i>Insertion.</i>
Levator Palati . . . .	Extremity of petrous bone; posterior and inferior aspect of Eustachian tube.	Middle line, where it is continuous with its fellow of the opposite side.
Tensor Palati (circumflexus palati).	Scaphoid fossa at base of internal pterygoid plate. Spinous process of sphenoid bone; anterior aspect of Eustachian tube.	Transverse ridge on the horizontal portion of the palate bone, and the aponeurosis of the velum.
Azygos Uvulæ . . . .	Spine of palate bone and adjoining tendinous structures.	Uvula.
Palato Glossus (constrictor isthmi faucium).	Soft palate continuous with its fellow of the opposite side.	Side of Tongue.
Palato-pharyngeus (constrictor isthmi faucium posterior).	Raphé of soft palate, and is continuous with its fellow of the opposite side.	Posterior border of the thyroid cartilage and side of the pharynx, as far back as the middle line.

*Nerve Supply.*—The levator palati and azygos uvulæ receive motor



FIG. 43.

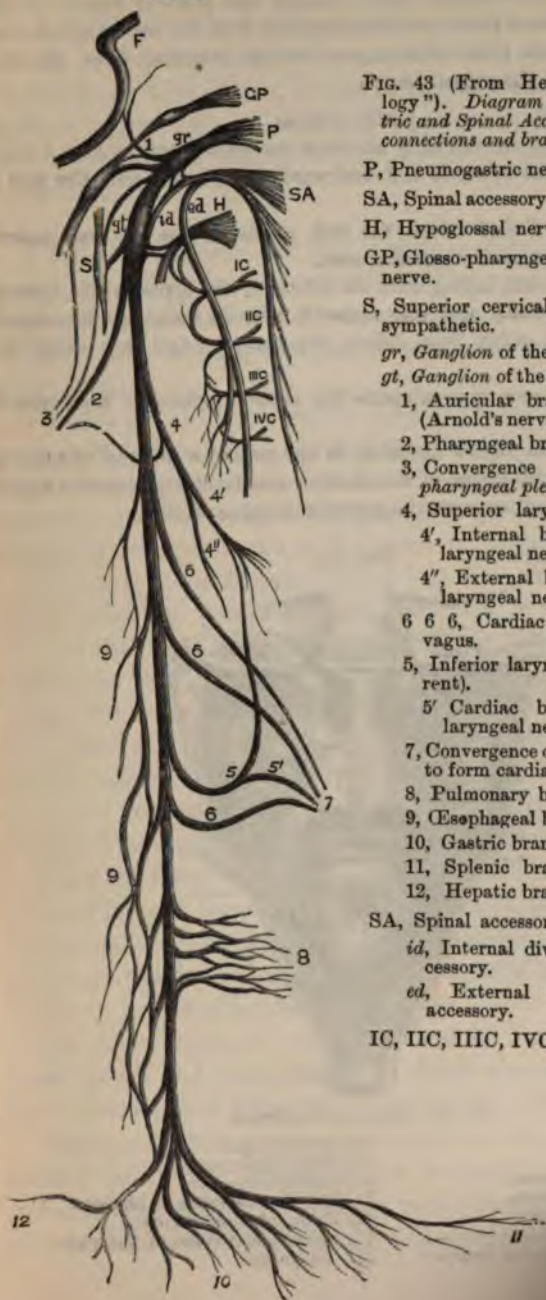


FIG. 43 (From Hermann's "Physiology"). Diagram of the Pneumogastric and Spinal Accessory Nerves, their connections and branches.

P, Pneumogastric nerve.

SA, Spinal accessory nerve.

H, Hypoglossal nerve.

GP, Glosso-pharyngeal nerve. F, Facial nerve.

S, Superior cervical ganglion of the sympathetic.

gr, Ganglion of the root of the vagus.

gt, Ganglion of the trunk of the vagus.

1, Auricular branch of the vagus (Arnold's nerve).

2, Pharyngeal branch.

3, Convergence of nerves to form pharyngeal plexus.

4, Superior laryngeal nerve.

4', Internal branch of superior laryngeal nerve.

4'', External branch of superior laryngeal nerve.

6 6 6, Cardiac branches of the vagus.

5, Inferior laryngeal nerve (recurrent).

5', Cardiac branch of inferior laryngeal nerve.

7, Convergence of branches of vagus to form cardiac plexuses.

8, Pulmonary branches.

9, Esophageal branches.

10, Gastric branches.

11, Splenic branches.

12, Hepatic branches.

SA, Spinal accessory nerve.

id, Internal division of spinal accessory.

ed, External division of spinal accessory.

IC, IIC, IIIC, IVC, Cervical nerves.

branches from the seventh nerve through the petrosal branch of the Vidian. The tensor palati receives branches from the otic ganglion; the palato-glossus and palato-pharyngeus receive branches from Meckel's ganglion and the pharyngeal plexus.

§ 263. *Actions.*

*The Azygos Uvulae* not only shortens the uvula, but renders it hard, and draws its point upwards and backwards towards the posterior wall of the pharynx.

*The Levator Palati* raises the soft palate, draws its free margin upwards, and elevates the palatine arch.

*The Tensor Palati*, acting with its fellow of the opposite side, spreads the palate out laterally, and also arches it with its concavity downwards. Taking its fixed point from below, the tensor palati will dilate the Eustachian tube.

*The Palato-glossus* approximates the anterior pillars of the fauces to the middle line.

*The Palato-pharyngeus* descends in the posterior pillar of the fauces, and, by opposing the action of the elevator muscle, it depresses the velum, and both lowers and narrows the posterior palatine arch.

FIG. 44.



FIG. 44. *Muscles of the Palate.*

- |                                     |                                      |
|-------------------------------------|--------------------------------------|
| 1, Septum narium.                   | 8, Azygos uvulae.                    |
| 2, Eustachian tube.                 | 9, Palato-pharyngeus.                |
| 3, Pterygoideus externus.           | 10, Stylo-pharyngeus.                |
| 4, Pterygoideus internus.           | 11, Middle constrictor of pharynx.   |
| 5, Levator palati mollis.           | 12, Palato-pharyngeus (cut).         |
| 6, Circumflexus palati.             | 13, Inferior constrictor of pharynx. |
| 7, Superior constrictor of pharynx. | 14, Œsophagus.                       |



§ 269. *Spasm of the Soft Palate and Eustachian Tube.*—Very little is known with respect to spasm of the muscles of the soft palate and of the associated muscular apparatus of the Eustachian tube. Noises in the ears can be induced in healthy individuals by intratubal application of the faradic or galvanic currents, caused by contraction of the tubal muscle (Weber-Liel).

§ 270. *Paralysis of the Soft Palate.*—Paralysis of the soft palate occurs in connection with bulbar paralysis and in the course of many diseases of the spinal cord. The partial paralysis which occurs in peripheral facial paralysis has already been mentioned.

The symptoms of paralysis of the soft palate differ according as the affection is partial or complete. Partial paralysis declares itself more especially by the abnormal position of the uvula and soft palate, while total paralysis gives rise to marked alterations of speech and in the power of deglutition.

*Paralysis of the Levator Palati.*—It has already been mentioned that the levator palati is paralysed in peripheral affections of the facial nerve when the lesion is situated above the geniculate ganglion. The velum on the paralysed side hangs loosely downwards, occupying a lower position during rest than the sound side. Tickling the uvula does not cause it to be arched upwards, it is only rendered tense transversely by the action of the tensor palati, and its posterior edge is drawn slightly downwards by the action of the palato-pharyngeus.

When the *levator palati* and *tensor palati* are simultaneously affected the soft palate hangs still deeper on that side, there is also lateral displacement of the velum owing to the unopposed action of the tensor palati of the opposite side; and reflex action on irritation is absent.

The disturbances of phonation and of deglutition, such as nasal speech and regurgitation of fluids through the nose, are more marked than when the levator palati is alone affected.

*Paralysis of the Azygos Uvulae* accompanies paralysis of the levator palati in disease of the seventh nerve above the geniculate ganglion. Unilateral paralysis of the azygos uvulae causes distortion of the uvula. The uvula occupies an oblique

position, with its point usually directed to the healthy side.\* In a case recorded by Sanders,† however, the uvula is described as "projecting somewhat forwards, and directed obliquely from left to right, the tip pointing to the right or paralysed side, its base being in the middle line, or perhaps drawn a little to the left or sound side." Four cases are described by Romberg, and one by Davaine, in which the uvula was arched with the convexity directed to the sound side, and the tip of the uvula to the paralysed side, and I have myself seen two well-marked instances of this distortion. It is difficult to account for the anomalous position occupied by the uvula in these cases. It has been suggested that the distortion to the paralysed side existed prior to the paralysis, as the uvula is not unfrequently found twisted to one side in healthy persons, but this could not have been the case in the observations of Romberg and Davaine, inasmuch as the uvula assumed a symmetrical position as the patient recovered from the paralysis. Sanders thinks that the drawing of the uvula towards the paralysed side is due to the action of the palato-pharyngeus exerting a greater effect on the uvula on the affected side than it can exert on the sound side; but this explanation is by no means satisfactory.

*Paralysis of the palato-pharyngeus* is recognised by the altered appearance of the isthmus of the fauces, the posterior pillars of the fauces being more widely separated from one another, and immovable. Duchenne believed that he had met with isolated paralysis of the palato-pharyngeus, where irritation still provoked shortening of the uvula, along with tension and elevation of the soft palate. The posterior pillars of the fauces were not approximated to one another, nor was the palate stretched downwards and backwards, so as to form a complete screen between the nasal and buccal portions of the pharynx. Speech and deglutition were unaffected.

Paralysis of all the muscles of the soft palate on both sides causes the palate to hang loose and flapping from the roof of the mouth, and its activity is not called forth during deep inspiration, or during the movements of deglutition and phonation. The speech has a strongly nasal character, or may be

\* Dr. Gairdner, *The Lancet*, Vol. I., 1861, p. 479.

† *Edinburgh Medical Journal*, Vol. XI., 1866, p. 144.



quite unintelligible, while fluids are ejected through the nose. Deglutition is rendered still more difficult when paralysis of the soft palate is associated, as frequently happens, with paralysis of the constrictors of the pharynx.

*Diagnosis.*—Paralysis of the muscles of the soft palate must be distinguished from immobility of the palate caused by mechanical and organic causes. The position assumed by the paralysed parts, the associated symptoms, and the history of the case are sufficient to prevent any mistakes being made.

*Prognosis.*—In diphtheritic paralysis of the soft palate the prognosis is favourable, while in the remaining forms the prognosis depends upon the disease on which it is dependent, and is, as a rule, unfavourable.

*Therapeutics.*—The treatment must generally be directed against the accompanying disease. The local treatment consists of the application of the faradic or galvanic currents to the muscles.

## (2) *Paralysis of the Muscles of the Pharynx and Œsophagus.*

The following table gives the muscles of the pharynx, their origins, and insertions :—

<i>Name.</i>	<i>Origin.</i>	<i>Insertion.</i>
Superior Constrictor	<ol style="list-style-type: none"> <li>1, The side of the tongue.</li> <li>2, Mucous membrane of the mouth.</li> <li>3, Mylo-hyoid ridge of jaw.</li> <li>4, Pterygo-maxillary ligament.</li> <li>5, Lower third of internal pterygoid plate.</li> </ol>	<ol style="list-style-type: none"> <li>1, Raphé of pharynx.</li> <li>2, Basilar process of occipital bone by means of pharyngeal aponeurosis</li> </ol>
Middle Constrictor	<ol style="list-style-type: none"> <li>1, Great cornu of os hyoides.</li> <li>2, Lesser cornu of os hyoides.</li> <li>3, Stylo-hyoid ligament.</li> </ol>	<ol style="list-style-type: none"> <li>1, Raphé of pharynx.</li> </ol>
Inferior Constrictor	<ol style="list-style-type: none"> <li>1, Cricoid cartilage.</li> <li>2, Oblique line of thyroid cartilage.</li> </ol>	<ol style="list-style-type: none"> <li>1, Middle line of pharynx.</li> </ol>
Stylo-pharyngeus	<ol style="list-style-type: none"> <li>1, Inner side of base of styloid process.</li> </ol>	<ol style="list-style-type: none"> <li>1, Posterior border of thyroid cartilage.</li> <li>2, Internal face of inferior constrictor.</li> </ol>
Palato-pharyngeus	<ol style="list-style-type: none"> <li>1, Raphé of soft palate, continuous with the corresponding muscle of the opposite side.</li> </ol>	<ol style="list-style-type: none"> <li>1, Inner surface of the pharynx.</li> <li>2, Posterior border of the thyroid cartilage.</li> </ol>
Salpingo-pharyngeus	<ol style="list-style-type: none"> <li>1, Lower border of Eustachian tube.</li> </ol>	<ol style="list-style-type: none"> <li>1, Unites with palato-pharyngeus.</li> </ol>

§ 271. *Actions.*

The constrictor muscles contract upon the morsel of food as soon as it is received by the pharynx and convey it downwards into the œsophagus. The stylo-pharyngei draw the pharynx upwards, and widen it laterally. The palato-pharyngei also draw it upwards, and with the aid of the uvula close the opening of the fauces. The salpingo-pharyngei are elevators of the upper part of the pharynx.

FIG. 45.

FIG. 45. *Muscles of the Pharynx* (from Heath).

- 1, Trachea.
- 2, Cricoid cartilage.
- 3, Crico-thyroid membrane.
- 4, Thyroid cartilage.
- 5, Thyro-hyoid membrane.
- 6, Os hyoides.
- 7, Stylo-hyoid ligament.
- 8, Œsophagus.
- 9, Inferior constrictor.
- 10, Middle constrictor.
- 11, Superior constrictor.
- 12, Stylo-pharyngeus, passing down between the superior and middle constrictor.
- 13, Fibrous bag of the pharynx seen above the constrictor.
- 14, Pterygo-maxillary ligament.
- 15, Buccinator.
- 16, Orbicularis oris.
- 17, Mylo-hyoideus.

FIG. 46.

FIG. 46. *Styloid Muscles and Muscles of the Tongue* (from Heath).

- 1, Temporal bone of the left side.
- 2, The right side of the lower jaw divided at its symphysis; the left side having been removed.
- 3, Tongue.
- 4, Genio-hyoideus.
- 5, Genio-hyo-glossus.
- 6, Hyo-glossus; its basio-glossus portion.
- 7, Its kerato-glossus portion.
- 8, Anterior fibres of the lingualis issuing from between the hyo-glossus and genio-hyo-glossus.
- 9, Stylo-glossus with part of the stylo-maxillary ligament.
- 10, Stylo-hyoideus.
- 11, Stylo-pharyngeus.
- 12, Os hyoides.
- 13, Thyro-hyoid membrane.
- 14, Thyroid cartilage.
- 15, Thyro-hyoid muscle arising from the oblique line of the thyroid cartilage.
- 16, Cricoid cartilage.
- 17, Crico-thyroid membrane.
- 18, Trachea.
- 19, Commencement of the œsophagus.



§ 272. *Dysphagia Paralytica.*

*Etiology.*—Paralysis of the muscles of the pharynx and of the muscular coat of the œsophagus is rare as the result of local peripheral disease. It results more frequently from basal affections of the brain, which produce compression upon the cranial nerves; and still more frequently from local affections of the pons and medulla oblongata. The paralysis of deglutition, which accompanies the terminal stages of progressive muscular atrophy, multiple sclerosis, and other nervous affections, as well as the dysphagia of typhus, and that which is always present during a prolonged death agony, probably originate in local disease of the pons and medulla. Dysphagia is not a frequent symptom in connection with apoplectic hemiplegia. Partial paralysis, generally limited to the superior constrictor, is a not unfrequent sequel of diphtheria and syphilitic affections, and is generally associated with paralysis of the soft palate and other muscles.

*Symptoms.*—The characteristic symptom of paralysis of the muscles of the pharynx is difficulty or impossibility of swallowing. When the paralysis is limited to the muscles of the pharynx, and does not implicate the facial or lingual muscles, or those of the soft palate, the loss of function first manifests itself during the act of swallowing. The morsel passes along the roof of the mouth and the back of the tongue, but remains on the root of the latter in the glosso-epiglottidean fossa, or even over the epiglottis; and must, on account of the dyspnœa to which it gives rise, be removed by means of the finger. Fluids run along the dorsum of the tongue, and pass readily into the larynx, giving rise to attacks of suffocating cough; while the patient instinctively makes strenuous efforts to pass the fluid over the epiglottis by throwing the head backwards, and by an endeavour to bring the root of the tongue as near as possible to the upper end of the œsophagus. If the paralysis be unilateral, the patient is only unable to swallow when the morsel of food happens to lodge on the paralysed side of the pharynx.

When there is only paresis of the muscles, or when the paralysis is limited to one or two of them, the power of swallowing

is only more or less diminished. If the paralysis be limited to the superior constrictor, fluids regurgitate through the nose during swallowing, inasmuch as contraction of this muscle is necessary in order to complete the division which the soft palate forms between the buccal and nasal portions of the pharynx during swallowing. This symptom is, of course, most pronounced when paralysis of the superior constrictor is associated with paralysis of the soft palate, as in the form which follows diphtheria.

*Paralysis of the Œsophagus* sometimes occurs as an isolated affection. The morsel of food in such a case passes from the pharynx into the œsophagus, but, owing to the failure of the peristaltic action of the latter, it remains fast in the cervical portion of the tube, or regurgitates into the cavity of the mouth. When it remains fast in the œsophagus it may produce compression of the larynx, and cause dyspnœa, and the other symptoms indicative of the presence of a foreign body. A large morsel of food, or solid substances which do not readily become reduced in volume, may still be swallowed as these make their way through the œsophagus by means of their own weight. This occurs with the greater facility because the tube becomes readily dilated when the muscular coat is paralysed.

Difficulty of swallowing may be present in various conditions of the œsophagus, and more especially in cases where there is a mechanical obstruction; but the diagnosis between these conditions and paralysis can readily be made by means of the sound.

*The Prognosis* is favourable in partial diphtheritic and syphilitic paralysis. The paralysis which arises from central causes is very unfavourable, owing to the gravity of the morbid processes underlying it; and complete paralysis, whatever its origin, may cause death more or less suddenly from arrest of the morsel of food in the œsophagus or air passages. Fatal attacks of lobular pneumonia are very liable to occur, owing to the passage of particles of food through the glottis, which find their way into the smaller ramifications of the bronchial tubes.

*Treatment.*—The treatment consists mainly in the local application of either the faradic or galvanic cur



affected muscles. \*In every case where there is complete paralysis, the patient must be fed by the stomach tube in order to avoid slow starvation and the accidents which are liable to occur during attempts at swallowing.

### (3) *Diseases of the Laryngeal Nerves.*

§ 273. The muscles of the larynx are eight in number, the five larger being muscles of the chorda vocalis and rima glottidis, the three smaller being muscles of the epiglottis. The following table gives the names of these muscles, with their origins and insertions, and the nerve by which they are supplied (Heath):—

<i>Name.</i>	<i>Origin.</i>	<i>Insertion.</i>	<i>Nerve.</i>
Crico-thyroideus.	Cricoid cartilage—Ring.	Thyroid cartilage, lower border and cornu.	External Laryngeal.
Arytænoideus.	Arytænoid cartilage, concave posterior surface.	Decussating fibres.	Superior and inferior laryngeal.
Crico-arytænoideus posticus.	Cricoid cartilage, posterior surface.	Arytænoid base, posterior outer angle.	Inferior laryngeal.
Crico-arytænoideus lateralis.	Cricoid cartilage, lateral border.	Arytænoid base, anterior outer angle.	Inferior laryngeal.
Thyro-arytænoideus.	Thyroid cartilage, receding angle.	Arytænoid, anterior surface.	Inferior laryngeal.
Thyro-epiglottideus.	External surface of sacculus laryngis.	Few fibres continued to side of epiglottis.	Inferior laryngeal.
Arytæno-epiglottideus, superior.	Apex of Arytænoid cartilage.	Side of epiglottis.	Inferior laryngeal.
Arytæno-epiglottideus, inferior.	Arytænoid cartilage above attachment of chorda vocalis.	Side of epiglottis.	Inferior laryngeal.

### § 274. *Actions.*

The muscles move the cartilages of the larynx upon one another in such a manner as to tighten and bring together or to separate the vocal cords, and thus to narrow or widen the aperture between them. The slit between the true vocal cords is called the *glottis vocalis*, and the interspace between the arytenoid cartilages the *glottis respiratoria*.

The *crico-thyroidei* draw the thyroid cartilage downwards on the cricoid cartilage, and thus tighten the vocal cords.

The *thyro-arytænoidei* are parallel to and attached to the same points as the vocal cords, and hence their contraction renders the vocal cords less tense. Some of the fibres, however, are inserted into different points of the vocal cords themselves, and consequently their contraction must confer different degrees of tension upon different parts of the cords.

The *crico-arytænoidei postici* swing the arytenoid cartilages outwards, so that both the *glottis respiratoria* and the *glottis vocalis* are converted

FIG. 47.

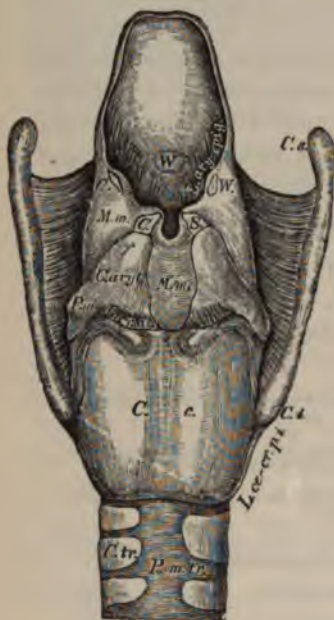


FIG. 47 (From Landois' "Physiologie").  
*Posterior View of the Larynx after Removal of the Muscles.*—*E.*, Epiglottis, with (*W.*) the cushion; *L.ar.ep.*, ligam. ary. epiglotticum; *M.m.*, Mucous membrane; *C.W.*, Wrisberg's cartilages; *C.S.*, Santorini's cartilages; *C.aryt.*, Arytaenoid cartilage; *C.c.*, Cricoid cartilage; *P.m.*, Muscular process to arytaenoid cartilage; *L.cr.ar.*, Crico-arytaen. ligament to superior cornu; *C.i.*, Inferior cornu of thyroid cartilage; *L.cr.p.i.*, Posterior inferior Kerato-cricoid ligament; *C.tr.*, Cartilages of the trachea; *P.m.tr.*, Membranous part of the trachea.

FIG. 48.

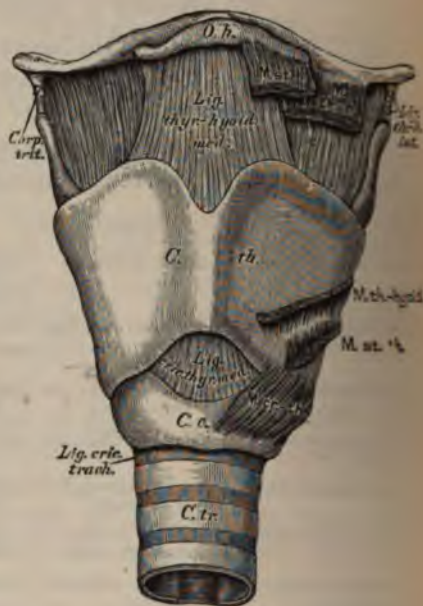


FIG. 48 (From Landois' "Physiologie").  
*Anterior View of the Larynx, showing the Ligaments and Attachments of the Muscles.*—*O.h.*, Hyoid bone; *C.th.*, Thyroid cartilage; *Corp. trit.*, Corpus triticeum; *Lig. thy.h.yoid. med.*, Ligamentum thyro-hyoideum medium; *Lig. th.h.lat.*, Ligament. crico-thyreoideum medium; *Lig. cric. trach.*, Ligam. crico-tracheale; *M.st.h.*, Sterno-hyoid muscle; *M.th.h.yoid.*, Thyro-hyoid muscle; *M.st.th.*, Sterno-thyroid muscle; *M.cr.th.*, Cricothyroid muscle.



FIG. 49.

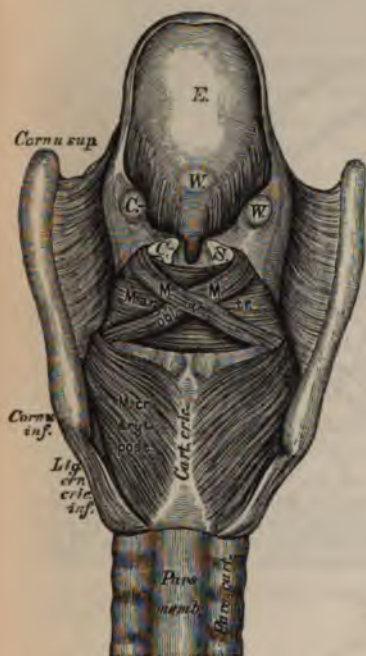


FIG. 49 (From Landois' "Physiologie"). *Posterior View of the Larynx, showing the Muscles.*—E., Epiglottis, with (W.) the cushion; C. W., Wrisberg's cartilage; C. S., Santorini's cartilage; Cart. cric., Cricoid cartilage; Cornu sup., Cornu inf., Superior and inferior cornua of the thyroid cartilage; M. ar. tr., Transverse arytenoid muscles; M. ar. obl., Oblique arytenoid muscles; M. cr. ar. p., Crico-arytenoidei postici muscles; Pars cart., Cartilaginous, and Pars mem., Membranous part of the trachea.

FIG. 50.

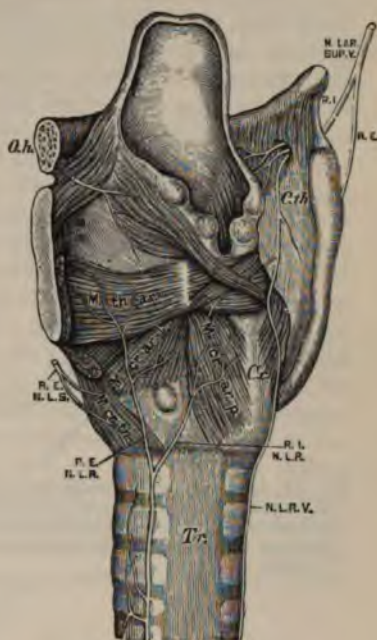


FIG. 50 (From Landois' "Physiologie"). *The Laryngeal Nerves.*—O. h., Hyoid bone; C. th., Thyroid cartilage; C. c., Cricoid cartilage; Tr., Trachea; M. cr. ar. p., Crico-arytenoideus posticus muscle; M. cr. ar. l., Crico-arytenoideus lateralis muscle; M. cr. th., Crico-thyroid muscle; N. l. ar. sup. v., Superior laryngeal nerve of the vagus, R. I., Internal branch, R. E., External branch; N. l. r. v., Recurrent laryngeal nerve, R. I. N. L. R., Internal branch; R. E. N. L. R., External branch of the recurrent laryngeal nerve.

into triangular spaces which, together, form a wide rhombic aperture (*Fig. 51, II II*).

The *crico-arytænoides laterales* are antagonistic to the *postici*, and bring the arytænoid cartilages back to their old position, and afterwards approximate them, thus narrowing the glottis (*Fig. 53, I I*).

FIG. 51.

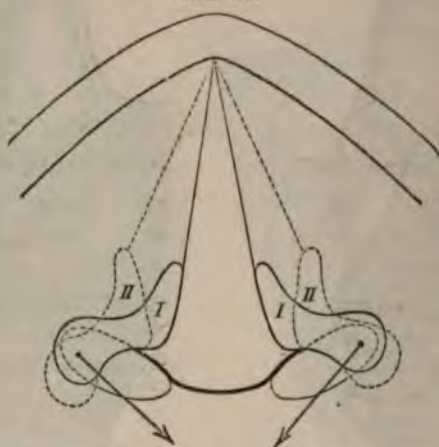


FIG. 51. *Schema of a Horizontal Section through the Larynx.*—*I I*, The position of the arytenoid cartilages and vocal cords during quiet breathing. The arrows indicate the direction in which the crico-arytænoides postici muscles act. *II II*, Position of the arytenoid cartilages in consequence of contraction of these muscles.

FIG 52.



FIG. 52 (From Landois' "Physiologie"). *Schema of a Horizontal Section through the Larynx, showing the action of the Arytænoid Muscles.*—*I I*, Position of the arytenoid cartilages during quiet breathing. The arrows show the direction of the action of the muscles. *II II*, Portion of the cartilages when the muscles are contracted.



The *arytænoidei* draw the arytenoid cartilages together at their apices and posterior edges. If they act in concert with the thyro-arytenoid muscles, both glottis vocalis and glottis respiratoria are closed, and respiration completely interrupted, as immediately before coughing (*Fig. 52, III*).

The three muscles of the epiglottis have been described as one muscle, under the name of thyro-arytæno-epiglottideus. Besides compressing the glands of the sacculus laryngis and altering the form of the cavity itself, this muscle, by its contraction, approximates the epiglottis and arytenoid cartilages, and thus assists in closing the glottis. It is important to remember that the position of the vocal cords during quiet breathing is not one indicative of complete passive equilibrium of all the laryngeal muscles. Immediately after death, when it may be presumed that complete passive equilibrium between the antagonistic muscles is established, the vocal cords are found much nearer the median line than they are during quiet breathing; hence it may be inferred that the latter position is maintained by a slight tonic action of the abductors of the glottis. The glottis assumes various forms according to circumstances, and it has been found useful to affix definite names to some of these. Passing from the middle line outwards these positions are: (1) Complete closure of the glottis (*Fig. 52, II II*), such as is produced by the combined action of the arytenoidei and crico-arytænoidei muscles; (2) *the cadaveric position*, so called from being the position in which the vocal cords are found in the dead body, this position being one in which the vocal cords are slightly removed from the median line, and consequently the glottis slightly open; (3) *the position of quiet breathing*, in which the glottis is more open than in the last position, but is still only moderately dilated (*Fig. 51, I I*); and (4) *the position of deep inspiration*, in which the glottis is widely dilated (*Fig. 51, II II*).

FIG. 53.



FIG. 53 (From Landois' "Physiologie"). Schema of Horizontal Section through the Larynx, illustrating the action of Crico-arytænoidei laterales Muscles.—II II, Position of the arytenoid cartilages during quiet breathing. The arrows show the direction in which the muscles act. I I, Position of the cartilages when the muscles are contracted.

§ 275. *Sensory Affections of the Larynx.*

The sensory disturbances may be divided into conditions of *anæsthesia*, *hyperæsthesia*, and *paræsthesia*.

*Anæsthesia* of the larynx is rare as an isolated affection, but is usually symptomatic of some other disease.

*Hyperæsthesia* of the laryngeal mucous membrane gives rise to excessive reflex actions on slight causes. A paroxysmal cough occurring in the female sex often depends upon excessive sensitiveness of the laryngeal mucous membrane.

*Paræsthesiæ* of the laryngeal mucous membrane are not unfrequently complained of. These may at times consist of a feeling as if a hair, fish-bone, or morsel of food had lodged in the larynx, while laryngoscopic examination shows the absence of a foreign body. Cases of this nature are most frequently met with in hysterical women.

§ 276. *Motor Affections of the Larynx.*

Motor disturbances of the larynx are much more important and frequent than sensory affections. The motor affections may be subdivided into *parakinesis*, *hyperkinesis*, and *akinesis*.

*The parakineses* consist of various inco-ordinatory movements of the laryngeal muscles, but not much is known with respect to them.

*The hyperkineses* consist of clonic or tonic spasm of the laryngeal muscles.

§ 277. *Clonic Spasm of the Laryngeal Muscles.*—In some cases the laryngeal muscles, or at least some of them, contract during attempts at phonation in such a way that the glottis is completely closed, and, no interspace being left between the vocal cords, complete aphonia results. Gerhardt mentions the case of a flutist who was not able to play his instrument without a continuous noise being caused in his throat. During this time it was found that the thyroid and cricoid cartilages were approximated, and the vibration of the vocal cords was distinctly perceptible on the surface. Involuntary movements also occurred in the arm which held the flute. These movements appear to have been produced



by an immoderate use of the instrument, and ceased after treatment with rest and bromide of potassium. Gerhardt thinks that this case is analogous to professional spasms in the hand; and, indeed, this patient had at a previous period suffered from writer's cramp.

§ 278. *Tonic Spasm of the Laryngeal Muscles (Aphonia Spastica. Laryngismus Stridulus).*

During spasm of the laryngeal muscles the action of the adductors predominate over that of their antagonists, and the glottis becomes completely closed.

*Etiology.*—The causes of spasm of the glottis may be central or peripheral. It occurs in hysteria and epilepsy, and indeed the laryngismus stridulus of children appears to be usually but a limited form of epileptic attack.

The peripheral causes may be direct or indirect. The direct causes consist of pressure on the recurrent or pneumogastric nerves by enlarged or tuberculous cervical and bronchial glands. Such pressure, however, as a rule, gives rise to paralysis.

The indirect peripheral causes produce spasm in a reflex manner. The inhalation of irritating gases and the entrance of morsels of food into the larynx are well known causes of reflex spasm. Spasm may also be produced by causes acting at a distance; examples of which may be found in the irritation of teething, or in the presence of parasites or other irritating substances in the elementary canal.

§ 279. *Spasm of the Glottis, or Internal Convulsions.*

*Etiology: Predisposing Causes.*—The causes which predispose to spasm of the glottis are generally the same as those which tend to produce eclampsia. Spasm of the glottis, however, is seldom observed in persons above one year of age, and usually attacks children between four and ten months. It is much more frequent in boys than girls; it attacks almost exclusively the children of the poorer classes, and badly-nourished cachectic or rachitic children. It also appears more frequently in cold weather, and especially in the month of March. All authors insist upon the preponderating influence of rickets

in the production of this disease. Out of fifty infants attacked by spasm of the glottis observed by Dr. Gee, forty-eight were rachitic.

*Occasional Causes.*—Cold is the most frequent exciting cause. It acts either directly on the terminations of the nerves of the laryngeal mucous membrane, or indirectly by producing a laryngo-tracheal catarrh. Fatigue of the muscles of the larynx caused by prolonged and excessive crying is sometimes an exciting cause of spasm of the glottis. When the disease is once established an attack may be provoked by the most various causes, as by examination of the throat, or attempts at deglutition, or sudden waking from sleep.

*Pathology.*—Spasm of the glottis appears to be entirely due to an irritable condition of the respiratory centre itself, or to a discharge from the cortex of the brain directed towards the medulla oblongata.

*Symptoms.*—The attack generally begins without premonitory symptom, and, according to West, occurs more frequently in the night than in the day. Spasm of the glottis manifests itself by a sudden attack of suffocation, respiration is suspended, the face becomes turgid, the countenance expresses great anxiety, the mouth is widely open as if to make a deep inspiration, the head is drawn backwards, the eyes fixed, the face becomes blue, and, in a word, the infant presents all the characteristics of commencing suffocation. The respiration may be suspended from two to twenty seconds, and the end of the attack is announced by a series of sonorous inspirations, as if air were drawn through a narrow reed. The last inspiration becomes longer and less sonorous, and then the respirations assume their natural rhythm. The sonorous inspiration is pathognomonic of spasm of the glottis, and, once heard, it is not easily forgot. The condition of expiration is variable. In some cases it is at first short and difficult, but gradually assumes its normal characters. In other cases the series of inspiratory convulsions is followed by a series of expiratory convulsions, short, sonorous, and jerky. In some exceptional cases, again, each sonorous inspiration is followed by a forced and noisy expiration, and only very rarely do the expiratory shocks constitute the initial phenomenon.



*Concomitant Symptoms.*—The functions of the body generally undergo a temporary disturbance. The pulse becomes accelerated, small, and sometimes scarcely appreciable, the action of the heart is tumultuous and irregular, and the chest is fixed. The veins of the neck and face become swelled, the skin is covered with a cold sweat, and the motions may be passed involuntarily. General epileptiform convulsions are only observed at the end of the attack, and not unless the spasm has been intense and prolonged. Sudden asphyxia, produced by the suspension of the respiration, is the principal cause of this complication. Spasm of the muscles of the extremities is, on the contrary, one of the symptoms habitually present during the attack, and it sometimes precedes the laryngeal spasm by some hours or days. The spasm appears to depend on the same general causes as that of the glottis. It is ordinarily limited to flexion of the hand and extension of the feet; but in some rare cases it invades the arms, thighs, and trunk.

*Course and Terminations.*—The disease may be limited to a single attack or to a series of paroxysms succeeding each other for some hours, and constituting a single attack; but this is the exception. Usually it is composed of a series of paroxysms coming on at irregular intervals during days or weeks. The following periods may be distinguished:—

(1) A period of augmentation, during which the attacks are rare, brief, and separated by intervals of perfect health. This period continues generally for some days, towards the end of which the attacks become more frequent and augment in intensity, and then the general health begins to suffer.

(2) A stationary period, during which the disease acquires its maximum intensity; the infant sometimes dies at this time, during a violent attack, from suffocation, or succumbs to marasmus or nervous exhaustion after repeated attacks.

(3) A period of decline, when the infant survives, during which the attacks lose their violence, become shorter, and are not so frequently repeated. The disease then terminates in complete recovery after a total duration of about from one week to two months, but relapses not unfrequently occur.

*Diagnosis.*—The diagnosis of spasm of the glottis is easy

when the physician happens to be present during the attack. The diagnosis is assisted by noticing the perfect health of the infant in the intervals between the attacks. Spasm of the glottis may be confounded with laryngismus stridulus, since both diseases have an intermittent and spasmodic character; but laryngismus appears in infants more than two years of age, the dyspnœa is more or less continuous, and there is a raucous cough.

*Prognosis.*—The mortality of spasm of the glottis is generally very high. Reid estimated it at 40 per cent; but Henoch does not make it so much. Rilliet and Barthez indicate as favourable circumstances brevity of the attacks, with a considerable interval between them; the presence of an expiration following each inspiration, the absence of signs of asphyxia, female sex, good hygienic and constitutional antecedents. The same authors regard the following as dangerous symptoms: (1) Great length and violence of the paroxysm, accompanied by cyanosis or great pallor of the face, and excessive smallness and feebleness of the pulse. (2) The repetition of the attack after a very short interval; that is, after half or three quarters of an hour, even if the first attack has not been a severe one. (3) Great nervous exhaustion after the attack.

*Treatment.*—During the attack plenty of fresh air should be allowed to circulate and all clothing should be removed from the throat. If the paroxysm be prolonged, the infant may be allowed to inhale a few drops of chloroform. If the infant is in a state of asphyxia, life must not be despaired of too soon, but every means for restoring suspended animation should be adopted, such as flapping the face with a wet towel, sprinkling cold water on the face, electricity, and artificial respiration.

In order to prevent a return of the attack tonic treatment must be adopted, as iron and cod-liver oil; the digestion must be carefully attended to, and good nourishing diet prescribed. The usual nervine remedies, as valerian, assafœtida, and zinc, have not been found of much use in the disease. Warm baths are frequently very soothing for infants, and the steam appears to exercise a soothing influence on the local spasm. During the attack a sponge wrung out of hot water may be applied to the throat.



§ 280. *Paralysis of the Laryngeal Branches of the Vagus*  
(*Paralysis of the Larynx—Aphonia Paralytica*).

*Etiology.*—Paralysis of the laryngeal muscles is generally of peripheral origin. The more usual causes are traumatic lesions, such as arise from gunshot injuries and surgical operations. Compression of the nerve by various tumours, such as swelled lymphatic glands in the neck, and more especially aneurism of the carotid and subclavian arteries, mediastinal tumours and enlarged bronchial glands, is the most frequent cause of paralysis. Other causes are, pleuritic adhesions and contractions about the apex of the lung, new growths, more especially cancer of the œsophagus and trachæa, which compress either the trunk of the vagus, or the recurrent laryngeal nerve. Paralysis of the laryngeal muscles is rare, as a result of disease or injury of the spinal accessory nerve prior to its union with the vagus.

Rheumatic paralysis as the result of exposure to a draught of cold air has also been observed, and paralysis may result from excess of functional activity, as after prolonged and loud speaking, screaming, and singing.

Paralysis of the vocal cords occurs as a sequel of acute diseases, as typhus, cholera, rheumatism, and diphtheria, as well as in syphilis, and in chronic lead and arsenical poisoning. Hysteria is one of the most frequent causes of this affection; while, on the other hand, aphonia is one of the most frequent symptoms of hysteria. Laryngeal paralysis from organic disease of the nerve centres is rare, but it sometimes occurs in the course of tabes dorsalis and in progressive cerebral paralyses. Unilateral paralysis of the vocal cords sometimes results from an attack of apoplectic hemiplegia.

*Symptoms.*—The larynx contains, on the one hand, the organs of voice and speech, and, on the other, it provides a free opening for the passage of air during respiration. Paralysis of the laryngeal muscles, therefore, gives rise to two prominent symptoms corresponding to the double function of the organ; the first consisting of disturbances of voice and speech, and the second of disturbances of respiration. When the vocal affection is the most prominent symptom, the disease has been called *phonetic paralysis*, and when the affection of

respiration predominates, it has been called *respiratory paralysis*; when there is marked disorder of both functions, the disease has been called *mixed laryngeal paralysis*.

Paralysis of the laryngeal muscles may be divided into unilateral or bilateral paralysis, according to its extent, and each of these may be subdivided into total or partial paralysis, according to its degree.

*Phonetic paralysis* occurs when the muscles which render tense and approximate the vocal cords are paralysed, and the affection of voice may vary from slight hoarseness to complete aphonia, according to the degree and extent of the paralysis.

*Respiratory paralysis*, on the other hand, occurs when the muscles which widen the aperture of the glottis—the crico-arytænoidei postici—are paralysed. In such cases the voice is unaffected, and even the disturbance of respiration may, during rest, be so slight as to be scarcely perceptible, although difficulty of respiration is readily induced on slight exertion; but at other times the affection gives rise to the most alarming inspiratory dyspnoea.

*Mixed paralysis* of the laryngeal muscles occurs when both the muscles which dilate the glottis and those which render tense and approximate the vocal cords are affected, and the disease is characterised by disturbances both of voice and of respiration.



FIG. 54 (From Landois' "Physiologie"). *Laryngoscopic appearances of the interior of the larynx.*—L, The root of the tongue; V. V., Glosso-epiglottidean ligament; E., The epiglottis; R, glottis; L. v., The true vocal cord; S. M., Opening into the sinus of Morgagni; L. v. s., The false vocal cords; S. S., The projection of the cartilages of Santorini; P., Pharynx wall; W. W., The cartilages of Wrisberg in the ary-epiglottidean ligament; S. p., The sinus piriformis.



The most important signs of paralysis of the laryngeal muscles are observed on laryngoscopic examination; and as the vocal cords are placed under the simplest condition in the mixed forms of paralysis, we shall describe these first.

*Mixed Paralyses.*--In complete bilateral paralysis of the laryngeal muscles the glottis assumes the cadaveric position, and remains immovable and unchanged during attempts at phonation. The relaxed cords are drawn somewhat downwards during inspiration, and pushed slightly upwards, and probably slightly removed from one another, during expiration (Wylie); but these slight movements are very different from the active movements caused by contracting muscles.

In *complete bilateral paralysis* the interference with respiration may be so slight as to escape notice during quietude; but dyspnœa is readily induced on slight exertion, and it is accompanied by stertorous or stridulous inspiration caused by the narrowed glottis, and the irregular manner in which the vocal cords vibrate. From the position of the vocal cords it is manifest that where the paralysis is complicated by a slight catarrh dyspnœa will be readily induced.

In *complete unilateral paralysis* of the laryngeal muscles the vocal cord of the corresponding side is motionless, its free edge is slightly removed from the middle line, and the vocal cord of the sound side alone vibrates during attempts at phonation.

FIG. 55.



FIG. 55. Laryngoscopic appearance of the larynx during quiet breathing.

FIG. 56.



FIG. 56. The laryngoscopic appearance of the larynx during vocalisation.

FIG. 57.



FIG. 57. The laryngoscopic appearance of the larynx during deep inspiration, showing the bifurcation of the trachea.

In *incomplete paralysis*, whether unilateral or bilateral, the power of excursion of the vocal cord is merely diminished and not entirely lost.

*Respiratory Paralysis.*—When the crico-arytænoidei postici are paralysed, the vocal cords assume the cadaveric position just as in complete paralysis of all the laryngeal muscles. In isolated paralysis of the abductors of the larynx, however, the approximation and parallelism of the vocal cords can still be accomplished, and the voice remains unaffected; but the glottis does not dilate during deep inspiration, and the cords cannot be separated beyond the cadaveric position. The respiratory function is at first only slightly interfered with, in a way exactly corresponding to that which has been described as occurring in complete paralysis of all the muscles. But in complete paralysis the contractile power of both the abductors and the adductors is abolished; while in paralysis of the crico-arytænoidei postici the abductors alone are paralysed, while the adductors remain active. The healthy adductors after a time undergo “paralytic contraction,” and consequently drag the vocal cords still further towards the middle line, so that the cadaveric position of the cords is much exceeded, and the glottis is almost completely closed. The glottis is now converted into a narrow slit, and becomes quite inadequate to carry on ordinary respiration.

When the paralysis is complete, the adductor muscles soon contract to such an extent as to give rise to the noisy breathing characteristic of croup. Owing to the marked constriction of the glottis respiration becomes laborious, the accessory muscles of inspiration are brought into action in order to overcome the obstruction, inspiration is prolonged and noisy, while expiration is comparatively easy and quick; the breathing is what has been described as the “forced costal type;” whilst, in consequence of the difference of atmospheric pressure above and below the constricted part, the larynx moves up and down considerably during each respiratory act.

On laryngeal examination it is seen that the vocal cords are approximated so that only a narrow linear chink is left between them, while the cords, instead of separating during forced inspiration, approach each other so closely as almost completely



to close the glottis. The cords are separated slightly during each expiratory act. During intonation the vocal cords and arytaenoid cartilages approach one another in a perfectly normal manner.

Incomplete bilateral paralysis may exist for a long time without giving rise to difficulty of breathing. It is probable that in many such cases the respiratory troubles continue so slight that they do not attract attention, and, owing to the entire absence of affection of voice, the patient may not be subjected to laryngoscopic examination, and the disease thus not detected. The affection also does not give rise to any very manifest symptoms when it is unilateral, but such a condition could doubtless be detected on laryngoscopic examination.

*Phonetic Paralysis.*—Paralysis of the muscles which render tense and approximate the vocal cords, gives rise to disorders of voice.

(1) *In bilateral paralysis* of the adductors the glottis is partially open, and both the arytaenoid cartilages and the vocal cords are immovable during attempts at phonation, the latter do not vibrate, and the glottis cannot be closed in coughing, on making an effort, or during deglutition; while there is complete aphonia.

(2) *In unilateral paralysis* of the adductors the vocal cord of the affected side is removed from the middle line, and cannot be approximated to its fellow. The affected vocal cord can only vibrate with its edge, and consequently the voice is feeble, and readily assumes the falsetto character.

(3) *Paralysis of the crico-thyroid* muscles occurs when the superior laryngeal nerve is implicated in disease, either directly or through the spinal accessory nerve. When these muscles are paralysed the vocal cords cannot be rendered tense; hence the voice becomes hoarse and deep, and the production of high notes is difficult or impossible. Disease of the superior laryngeal nerve also paralyses the thyro-arytaeno-epiglottidei, and the epiglottis is consequently drawn towards the tongue, so that during deglutition it is not depressed over the aperture of the glottis; hence food and drink obtain entrance into the larynx, and give rise to distressing symptoms. In paralysis of the tensors of the vocal cords the glottis closes completely

during strong expiratory efforts, as in coughing, and the aryæ-noid cartilages are quite movable during attempts at phonation.

(4) *In paralysis of the thyro-arytænoidæi* the ligamentous part of the glottis remains open, while juxtaposition of the arytænoid cartilages takes place.

(5) *In paralysis of the crico-arytænoidæi lateralis* the glottis remains open in the form of a tolerably broad ellipse.

(6) *In paralysis of the arytænoidæi* the ligamentous portion closes almost completely, while the interspace between the ary-tænoid cartilages remains open.

Although loss of voice is present in paralysis of all the tensor and constrictor muscles, yet it is most marked in the cases where the interspace between the cartilages remains open; while the voice may be very little affected in the cases where the cartilages close and the ligamentous portion remains open. Under the former circumstances the blast of air escapes through the patent glottis respiratoria, and the cords are not set in vibration; but under the latter, the blast of air must pass through the glottis vocalis, and a certain degree of vibration of the cords will be caused.

In peripheral laryngeal paralysis, due to compression or injury to the recurrent nerve, the muscles, like others under similar conditions, lose their faradic and galvanic contractility.

(7) *Rheumatic laryngeal paralysis* is, as a rule, unilateral, and may be complete or incomplete; occasionally it is bilateral. When the paralysis is incomplete, the thyro-arytænoidæi are affected by preference; and in paralysis of these muscles, as already remarked, the ligamentous portion of the glottis does not close completely. Rheumatic paralysis is frequently associated with catarrhal conditions of the larynx, but how far any causal relation obtains between them is not known.

(8) *Hysterical aphonia* appears generally to result from paralysis of the arytænoid muscles; so that while the ligamentous portion of the glottis closes, the interspace between the cartilages remains open. This form of aphonia occurs in paroxysms, which sometimes come on at regular periods of the day; and during the interval the laryngoscopic appearances are quite normal.

*The Diagnosis* depends entirely on the appearances presented



by the glottis on laryngoscopic examination, although valuable confirmatory evidence is afforded by the general symptoms.

*The Prognosis* depends upon the nature of the cause. It is most favourable in hysterical and rheumatic paralysis, and in that which occurs after severe exertion of the voice, and as a sequel of acute infectious diseases. When the paralysis is caused by compression or injury of the nerve, the prognosis is unfavourable, and it is equally unfavourable in paralysis of organic disease of the nerve centres.

*Treatment.*—The treatment must vary according to the cause of the affection, and in severe cases of laryngeal paralysis, due to compression of the recurrent nerves by aneurisms and other tumours, the treatment must be directed to the primary disease, and this, unfortunately, is in most cases of very little avail as far as the laryngeal symptoms are concerned. In most cases of laryngeal paralysis the best direct treatment consists in the local application of the faradic current. One of the electrodes may be passed by the aid of the laryngoscope into the larynx, while the other is placed on some part of the surface. The instruments devised by Duchenne and by Morel Mackenzie answer very well for the purpose, but this method of application is somewhat troublesome, and requires a certain amount of special skill not possessed by the majority of physicians. Except in very obstinate cases the percutaneous application of the faradic or galvanic currents is amply sufficient, so that the trouble of passing the electrode into the larynx is avoided. In hysterical aphonia, indeed, the application of the faradic brush over the surface of the larynx, so as to cause pain, is probably more efficacious than even intralaryngeal faradisation.

The subcutaneous injection of strychnia has been found useful by Waldenburg and others in rheumatic and catarrhal laryngeal paralysis, and in that occurring after diphtheria.

#### § 281. *Neuroses of the Pulmonary Plexus.*

*Spasm of the Bronchioles (Asthma bronchiale seu nervosum).* Nervous asthma consists of paroxysmal attacks of difficulty of breathing caused by general contraction of the smaller bronchial tubes.

*Etiology.*—Asthma is often inherited, and frequently descends

directly from parents to children. One member of a family may suffer from asthma, while others suffer from epilepsy and allied affections. Males are twice as frequently affected as females. The first attack generally occurs prior to the age of ten, although it may appear at any age from birth to extreme old age.

The exciting causes may be direct or indirect. The direct causes act on the mucous membrane of the bronchial tubes. The most usual of these are the inhalation of smoke, dust, or irritating gases; the smell of cats, dogs, horses, or other animals; the scent of the privet, rose, and other flowers; the emanations from new-mown hay and powdered ipecacuanha. Change of locality has a very curious influence on the disease. Some asthmatics can live a comfortable life in the most crowded thoroughfares of towns, who are subject to severe paroxysms of dyspnœa in pure country air. Some suffer most in high altitudes, others at a low elevation, some in dry, others in a moist atmosphere, and some can live with tolerable comfort on one side of a street and suffer greatly on the opposite side. I have seen a boy four years of age who had his first attack on being transferred from London to a country village in Yorkshire, far removed from the smoke of factories. He had a severe paroxysm on each of three consecutive nights in which he slept in one bedroom; but on being transferred on the third night to an adjoining bedroom the paroxysm ceased, and he had only one attack during the following six months. On this occasion he was sent to sleep in the first bedroom, but a severe paroxysm soon developing he was obliged to be removed, when the dyspnœa immediately ceased.

The indirect causes are certain articles of diet, which differ almost for each patient, distention of the stomach, constipation, and violent emotions.

*Symptoms.*—The asthmatic attack is often preceded by certain premonitory symptoms, which vary in different cases. The most usual symptom consists of a feeling of constriction across the chest with a slight tendency to wheeze, but at times it consists of flatulence, depression, or even unusual buoyancy of spirits. One asthmatic patient told me that he knew a severe attack was about to begin when he passed an abundant quantity of clear limpid urine. The symptoms



of the paroxysm are those of intense dyspnoea, and they are usually well described in works devoted to diseases of the chest, so that it is unnecessary to describe them in detail in this place. The patient is probably roused at from two to four o'clock in the morning with an intense feeling of suffocation. During the paroxysm the breathing is slow and laboured and effected with the most violent effort. The patient sits with head thrown back, dilated nostrils, and mouth widely open. The accessory muscles of inspiration are thrown into energetic action, and the patient generally grasps some fixed object so as to give them increased purchase. The patient bears an expression of deep anxiety; the surface is pale and ghastly; the hands and feet cold and livid; the body bathed in sweat; the eyes are congested and protruding; the pulse is small, feeble, and often irregular. The lungs become expanded and hyperresonant on percussion, the diaphragm is depressed, and the liver and spleen displaced downwards; and on auscultation the normal respiratory murmur is completely replaced by loud sibilant ronchi.

The duration of a single paroxysm is comparatively brief, but a series of them may extend over a few weeks.

*Prognosis.*—So far as any immediate danger to life is concerned the prognosis is favourable; but as a rule it recurs at regular intervals of time during the remainder of life. When the disease begins in infancy it often disappears during the adult period of life, but when it comes on late in life it is usually permanent.

*Pathology.*—The symptoms are caused by spasmodic contraction of the muscular tissue of the bronchial tubes. This spasm may be excited either by direct irritation of the trunk of the vagus, or in a reflex manner by irritation of the sensory nerves.

*Treatment.*—The treatment during the attack must be directed to allay the distressing symptoms. All the exciting causes, whether acting directly on the bronchi, or indirectly through the stomach uterus or other organs, must, if possible, be removed. The most useful direct remedies during the attack are tartar emetic, ipecacuanha in emetic doses; while tobacco, lobelia inflata, datura stramonium or datura tatula may be used as cigarettes, or in the form of tincture or extract.

Belladonna, conium, or hyoscyamus may be given in strong coffee; alcohol, and nitre paper burnt in the apartment, are useful and often convenient remedies; but in severe paroxysms the greatest relief is obtained from the inhalation of chloroform or ether.

In the interval between the attacks care must be taken to avoid all the exciting causes of the affection. The patient must be allowed to select his own residence, as it is not possible beforehand to tell what kind of air and climate will best suit him. The greatest care must be taken in the regulation of the diet, and in maintaining the surface of the body from being chilled by wearing flannel next the skin.

#### § 282. *Neuroses of the Trunk of the Vagus.*

The trunk of the vagus is liable to be injured by various causes, such as gunshot wounds, section, excision or ligature of the nerve during surgical operations for the extirpation of tumours, or ligature of the carotid artery; and compression or secondary implication of the vagus from tumours of the lymphatic glands, abscesses in the neck, aneurism of the larger arterial trunks in the chest and neck.

In the majority of cases the injury is limited to one side, and may occur either above or below the point of origin of the recurrent laryngeal. The symptoms correspond in the majority of cases to those which are observed in the lower animals after experimental injury. When the phenomena of irritation predominate, the action of the heart is rendered slow and irregular, and the symptoms of angina may be present.

More frequently, however, the symptoms are those of paralysis of the vagus. Passing over the symptoms of laryngeal paralysis, which have been described already, paralysis of the vagus, either unilateral or bilateral, causes increase in the frequency of the pulse, which may beat permanently at the rate of 120—160 per minute, and the radial pulse becomes small and scarcely perceptible, and the arterial tension is diminished.

The alteration of the pulse is a striking symptom of slow growing mediastinal tumours, and may, indeed, for a long time be the only symptom by which the presence of the tumour can be suspected. In such cases the action of the heart does



not appear to be rendered slow by the use of digitalis. In the majority of cases the symptoms caused by mechanical compression of the lungs and surrounding textures are present, and then the diagnosis is much facilitated. In pure compression of the vagus, death may result from syncope caused by irritation of the inhibitory fibres of the vagus.

§ 283. *Neuroses of the Cardiac and Gastric Plexuses.*

The part which the vagus takes in the neuroses of the cardiac and gastric plexuses cannot as yet be separated from the action of the sympathetic; hence it will be as well to deal with these plexuses when the diseases of the sympathetic system of nerves are under consideration.

(III.)—DISEASES OF THE SPINAL ACCESSORY NERVE.

The spinal accessory nerve subdivides into two branches—an external derived from the spinal cord, and an internal branch derived from the medulla oblongata. The external branch is distributed to the sterno-cleido-mastoid, and trapezius muscles, which receive branches also from the cervical plexus; the internal branch blends with the trunk of the pneumogastric nerve, and supplies the muscles of the larynx through the recurrent laryngeal nerve. The spinal accessory being a purely motor nerve, its diseases ought probably to have been described in the preceding chapter; but inasmuch as it belongs to what Meynert calls the “mixed lateral system” of nerves, and is closely related anatomically to the vagus, I have determined to place the affections of this nerve amongst those of the mixed cranial nerves.

§ 284. *Spasm in the Region of the External Branch of the Spinal Accessory. Wry-neck. Spasmodic Wry-neck (Caput Obstipum Spasticum).*

*Etiology.*—The causes of the disease are often obscure. It may come on suddenly during the night, and without any assignable cause; but more commonly the onset is gradual, and often so insidious that the real nature of the malady is at first overlooked. Brodie mentions the case of a woman in whom the spasmodic affection alternated with insanity. The rela-

tives of those suffering from wry-neck are not unfrequently subject to hysteria and other nervous affections.

The male sex is rather more frequently affected than the female, and the disease is generally an affection of adult life. The most usual exciting causes are excessive exertion, violent emotions, exposure to cold, drunkenness, and poverty. It may be caused by reflex irritation of remote organs, such as the intestinal canal and uterus.

*Symptoms.*—*Wry-neck* may be, like facial spasm, either tonic or clonic; but, unlike what occurs in the latter disease, the tonic is much more common than the clonic form of the former.

(1) The *tonic* form (*caput obstipum spasticum*) is almost exclusively confined to one of the sterno-cleido-mastoid muscles, but a part of the trapezius is often affected. This form of the disease being always unilateral the contracted muscle, from the obliquity of its direction, rotates the head so that the occiput is approximated to the shoulder of the affected side, the ear of the same side drawn towards the clavicle, and the chin turned upwards and away from the opposite shoulder. In chronic cases the cervical region of the vertebral column becomes the seat of

FIG. 58.



FIG. 58 (From Duchenne). *Spasm of the Trapezius.*



permanent curvature, the convexity of which is directed to the sound side, while there is a compensatory curve in the dorsal and lumbar regions. When the trapezius alone is the seat of spasm the head is drawn strongly backwards and inclined to the affected side, there is no turning of the chin, the point of the shoulder is elevated, and on any attempt being made to bend the head forwards the muscle becomes tense and painful.

(2) The *clonic* form of spasm of the muscles of the neck may be either unilateral or bilateral. Sometimes, indeed, the unilateral variety invades both sides, but in such a case the muscles on each side contract alternately or quite irregularly; while in the true bilateral variety the affected muscles on both sides act in concert.

The disease generally begins with uneasiness in the neck, extending from the back of the neck or the occiput to one of the shoulders. It is soon noticed by the patient or his friends that the head is not straight, and as the disease advances the uneasiness amounts to pain, which may occasionally be very severe, but is generally of a dull aching character, seated in the course or at the insertion of the muscle. The movements of the head will of course depend upon the muscles attacked; but in the usual variety, where one of the sterno-cleido-mastoid muscles is affected, it is rotated obliquely to one side by a succession of jerks in such a manner that the occiput is turned towards the shoulder and depressed, and the chin is elevated in the opposite direction. The muscle on the side to which the head is drawn down is found hard and contracted, and frequently hypertrophied. When the trapezius is affected, the head is bent back and the shoulder raised as has already been described. At the end of a short time, generally a few seconds, the muscle relaxes and the head returns to the normal position; but this is soon followed by a second contraction and a second rotation. In the beginning of the disease there is a considerable interval between each contraction, but as it advances this interval is so shortened that there may be 22 (Bell) or 30 (Hasse) contractions in the minute. Romberg counted 11 oscillations of the head in fifteen seconds in a young girl affected by the disease. In an early stage of the affection the patient may arrest the spasm by a voluntary

effort, and Bell mentions a case where the patient, when the paroxysm was at its worst, was able by volition to relax the muscle, but only for a short time. The spasm may also be arrested by fixing the head and neck; and with this view the patient frequently supports the head with both hands or places it against a resisting object, as a wall; and it may also cease if the attention be strongly attracted. On the other hand, the spasms are increased by everything that lowers the general health, by emotional excitement, and physical exertion.

In a case of moderate intensity the patient is able to counteract the spasm by a voluntary contraction of the muscles of the opposite side; and from the continual antagonism of the involuntary and voluntary actions during waking hours, the head presents a series of oscillations, in which the chin is deviated to the opposite side by the former, and immediately drawn back to the middle line by the latter, so that the head on the whole is maintained in a central position. But when the disease has lasted for a longer period, the voluntary is overpowered by the spasmodic action, so that the patient cannot (without extraneous aid, as by pulling it with the hands) bring the head into a central position, and it is consequently habitually twisted to one side. Even under these circumstances the patient may by a strong voluntary effort bring the chin to the middle line, but the effort causes distress, and cannot be maintained.

Patients are distressed by wakefulness, aggravated by the spasmodic movements of the head against the pillow; but when sleep is coming on they usually feel the movements become gradually less and less, and the spasm ceases entirely during sleep. This rule, however, is not without its exceptions, since Bell mentions the case of a patient who had perpetual rolling of the head both night and day.

The disease is very rarely confined to the muscles supplied by the spinal accessory nerve; indeed, there is some reason to believe that the deeply-seated muscles at the back of the neck—the *splenii* and *obliqui capitis*—which receive their motor power from the superior cervical nerve, are frequently the first to be affected, and they are at least generally implicated. When the *scaleni* are affected, Romberg has observed anæsthesia and oedema of the corresponding arm caused by compression of the brachial



plexus and veins. The facial is also very frequently involved, producing various distortions of the face; the motor branch of the trigeminus causing masticatory spasm; and in severe cases the disease extends to the other cervical nerves and to the brachial plexus, resulting in various spasmodic movements of the arm, hand, and fingers, closely simulating chorea. Two cases came under my notice in which spasmodic action of the muscles of the face and neck was associated with spasm of some of the intercostal muscles of the same side, the action of the latter being specially apparent in the axillary region; and Bell says of a patient, "while these very severe fits last, which is for about a minute each time, his breathing is performed with difficulty, and he gasps as if he were suffocating." This variety of the disease is usually confined to the one side of the body, but occasionally both sides are affected. When the two sterno-cleido-mastoids are implicated, the head is rotated first to the one side and after a time to the other, according as the action of the one or of the other predominates; and occasionally the two may happen to contract simultaneously, in which case the head is bent forwards and the chin drawn to the sternum. In very violent cases the disease extends even to the muscles of the lower extremities, so that, according to the description of Bell, "the whole body partakes of the tremor." Occasionally difficulty of deglutition and huskiness of voice have been noticed as symptoms, owing no doubt to the extension of the spasm to the muscles respectively concerned in those functions. The faradic irritability of the affected muscles appears to be increased, and an interrupted current, which causes no pain on the healthy side, may cause great pain when passed through the affected muscles. When a constant current of moderate intensity is passed through the contracting muscles the spasm relaxes, but it returns immediately, or soon afterwards, when the current is withdrawn.

Points of arrest are frequently observed in the course of the nerve or over the affected muscles.

(3) The *bilateral* clonic variety (Eclampsia nutans—Salaam convulsion) is almost exclusively confined to children from the first period of dentition to puberty. The disease comes on in paroxysms, each of which lasts only from a few seconds to some

minutes, but generally recurs once or two or three times in the course of the day. Sometimes, however, there may be as many as six or ten attacks in an hour, while at other times several days may intervene between them. During the attack the body and head are bent slightly forward, and this is followed by almost instantaneous relaxation, to be succeeded after an interval of a few seconds by a second bowing of the head; and so on until the paroxysm ceases. The bowing of the body and head may be repeated as often as twenty, fifty, or a hundred times during the attack. Facial spasm, blepharospasm, strabismus, or a slight convulsive movement of one or other arm, or of one of the lower extremities, is often associated with the spasmodic action of the muscles of the neck, and attacks of general convulsions frequently intervene, so that the case becomes one of ordinary epilepsy. During the attack the child seems bewildered, but there is not complete loss of consciousness; and as soon as the movements cease, the patient may be quite bright and happy. The attack may sometimes be followed by exhaustion and drowsiness.

*Diagnosis.*—The “stiff neck” produced by exposure to cold may be mistaken for the tonic form of torticollis. In the former case, any endeavour to move the muscle causes great pain, and usually the affection is very temporary in its duration.

A tonic spasmodic affection of the muscles of the neck may be the first symptom to reveal inflammation of the cervical vertebræ or spinal meningitis; but in such cases there is marked tenderness on pressure of the spinous processes, and some fulness or hardness around or behind the vertebral column; and if the disease progresses, these symptoms are succeeded by deformity of the superior cervical region of the vertebral column and paralysis.

The position of the head may also be affected by various tumours and by extensive cicatrices, but these cases are not likely to be confounded with genuine torticollis. Torticollis is sometimes a symptom of organic diseases of the brain, accompanied by hemiplegia; but the existence of the paralysis is sufficient to distinguish such cases from the local disease.

*Morbid Anatomy and Physiology.*—No appreciable lesion of that part of the nervous system with which the disease is



necessarily associated has hitherto been detected. According to Volkmann, irritation of the spinal accessory nerve in its passage through the foramen lacerum causes contraction of the sterno-cleido-mastoid and trapezius muscles in a recently killed animal; and Brown-Séquard found that torticollis resulted from injury to certain muscles, the olivary body, or the auditory nerve. The disease is analogous to writers' cramp and facial spasm, but the pathology of all these spasmodic affections must be left for future investigation. The bilateral clonic variety sometimes results from reflex irritation, as that caused by teething, and it may cease spontaneously after the first dentition. The tendency of these cases, however, is to pass into confirmed epilepsy, ending generally in great impairment of the intellect, a course which is often followed by other partial convulsions.

*Prognosis.*—When torticollis is of three or four months' duration it becomes a most obstinate malady. If the unilateral clonic form be of moderate intensity it may gradually improve and yield to treatment, but it almost invariably recurs, and in the end resists every effort made to remove it. The general health is not necessarily impaired, except in those severe cases where the patient is worn out by the constant agitation and want of sleep.

The tendency of the bilateral variety to pass into genuine epilepsy and idiotcy renders the prognosis much more grave.

*Treatment.*—For the tonic variety, myotomy and the subsequent application of a suitable mechanism for maintaining the head in a straight position are the proper remedies. In the bilateral clonic form all possible sources of irritation must be carefully searched for and removed, and if the convulsions still continue they ought to be treated from the first like genuine epilepsy, efficient doses of the bromide of potassium being the most promising means at present known.

The treatment of the clonic form has not hitherto been attended with much success. Romberg obtained a satisfactory result by progressively increasing doses of sulphate of zinc. Hammond reports success with large doses of the bromide of potassium, and it is probable that the bromide of zinc in gradually increasing doses might be found useful. The sub-

cutaneous injection of morphia and of atropia have each been followed by great benefit.

Moritz Meyer has been successful with electricity, but in the hands of others the use of this agent has not been followed by such brilliant results, although a certain amount of benefit has followed. The best method of employing electricity is to apply a moderate continuous current to the muscles affected with spasm, and a faradic current to their antagonists. Permanent compression by a suitable mechanism over the points of arrest has led to cure. In the case of a policeman kindly sent to me by Dr. Dacre Fox, and in whom spasmodic torticollis suddenly developed after exposure to wet and cold, a suitable mechanism was applied for producing pressure on the "point of arrest," under Dr. Hardie's superintendence. The instrument gave great relief to the patient, but after treatment for six months the spasmodic symptoms were not decidedly improved. The patient then left Manchester, and I heard through Dr. Fox that he continued to wear the instrument, and that there was no abatement of the spasm until about six months afterwards, or twelve from the onset of the disease. About that time he went to bed one night, affected as usual, and woke up in the morning finding himself free from spasm, and had no return of the symptoms at the date of the report, some weeks subsequently. I do not know whether the symptoms recurred, and it is of course impossible to determine what share, if any, the treatment had in contributing to the favourable termination of the case.

Stretching of the nerve has also been tried, but the operation has not hitherto been successful. Dr. J. Bennet records a case successfully treated by division of the nerve after stretching had failed. Division of the branches of the spinal accessory nerve was attempted by Dr. Bujalsky, as reported by Stromeyer, but it was not followed by any permanent results. Favourable results have been obtained by division of the affected muscles, an operation which succeeded in the hands of Stromeyer; but was utterly ineffectual in a case where Dieffenbach repeatedly divided the sterno-cleido-mastoid, which was affected by intense spasm.

Various mechanical contrivances in order to force and main-



tain the head in position have been employed, but, although these may be of temporary advantage, they cannot be borne habitually by patients suffering from confirmed torticollis.

§ 285. *Paralysis of the External Branch of the Spinal Accessory Nerve.*—Paralysis of the sterno-cleido-mastoid and trapezius may co-exist, or either may occur alone.

*Etiology.*—Paralysis in the region of distribution of the spinal accessory is generally of peripheral origin, and results from compression of the nerve by tumours, enlarged lymphatic glands, abscesses, or disease of the bones of the skull, or traumatic injuries to the nerve. Paralysis may also result from exposure to cold, or from neuritis in whatever way caused. The muscles supplied by the spinal accessory may also be paralysed and atrophied in progressive muscular atrophy.

*Symptoms.*—Paralysis of the sterno-cleido-mastoid and trapezius muscles may be unilateral or bilateral; while the muscles may be separately or simultaneously affected.

In unilateral paralysis of the sterno-cleido-mastoid the head is held in an oblique position, the chin is elevated and turned towards the affected side, the prominence of the healthy muscle on its movements being resisted is absent, the head can only be moved in the opposite direction with difficulty and by the aid of other muscles, although passive movements can be readily performed. In chronic cases contraction of the healthy muscle occurs, and causes persistent obliquity of the position of the head.

With bilateral paralysis of the sterno-cleido-mastoid muscles the head is held straight, but rotation of the head, when the chin is elevated, can only be performed with difficulty. The prominence of the muscles is absent, the neck looks wasted, and, if atrophy be present, a slight depression is produced between the mastoid process and the sternum.

In paralysis of the trapezius the scapula of the affected side is drawn somewhat downwards and forwards, its inner border is separated from the vertebral column and placed obliquely, so that the inferior angle becomes relatively near to the vertebral column. The acromion process falls downwards and forwards,

partly from the weight of the arms and partly from the antagonistic action of the rhomboideus and levator anguli scapulæ; hence the clavicle projects, the supra-clavicular fossa is deeper than natural, and the posterior and superior angle of the scapula can be felt with unusual distinctness. The trapezius derives its nervous supply from various sources, hence partial paralysis of the muscle is not uncommon, and in such cases the position of the scapula differs to some extent according as the upper, middle, or lower third of the muscle is paralysed. The upper third of the trapezius draws the acromion upwards and backwards, and paralysis of this portion of the muscle renders elevation of the arm above the horizontal line difficult.

If the paralysis be bilateral, the symptoms are present on both sides, both shoulder blades fall outwards and forwards, the head readily sinks on the chest, and some difficulty is experienced in maintaining it in an upright and straight position.

If both the sterno-cleido-mastoid and trapezius be simultaneously affected, the symptoms of the separate paralyses are combined, and the internal branch of the spinal accessory is not unfrequently implicated.

*Treatment.*—An endeavour must be made to remove the cause, and subsequent electrical treatment is of the greatest importance. Deformities produced by secondary contractions must be removed by the aid of active and passive gymnastics, tenotomy, and various mechanical appliances.



## CHAPTER VI.

## DISEASES OF THE CERVICAL AND BRACHIAL PLEXUSES.

## (I.)—DISEASES OF THE CERVICAL PLEXUS.

§ 286. *Cervico-Occipital Neuralgia.*

CERVICO-OCCIPITAL NEURALGIA has its seat in the region to which the sensory fibres of the four upper cervical nerves are distributed. The cutaneous nerves of the cervical plexus are:—

(1) The great occipital nerve, distributed to the whole of the occipital and posterior parietal regions as far as the vertex; (2) the small occipital nerve, which is distributed over the side of the back part of the head as far forwards as the ear; (3) the great auricular nerve, which supplies the face, the parotid region, and the back of the external ear; (4) the inferior subcutaneous nerve of the neck, which is distributed over the anterior region of the neck, the chin, and the side of the cheek; and (5) the supra-clavicular nerves, which ramify throughout the clavicular and upper thoracic regions and the lower part of the neck.

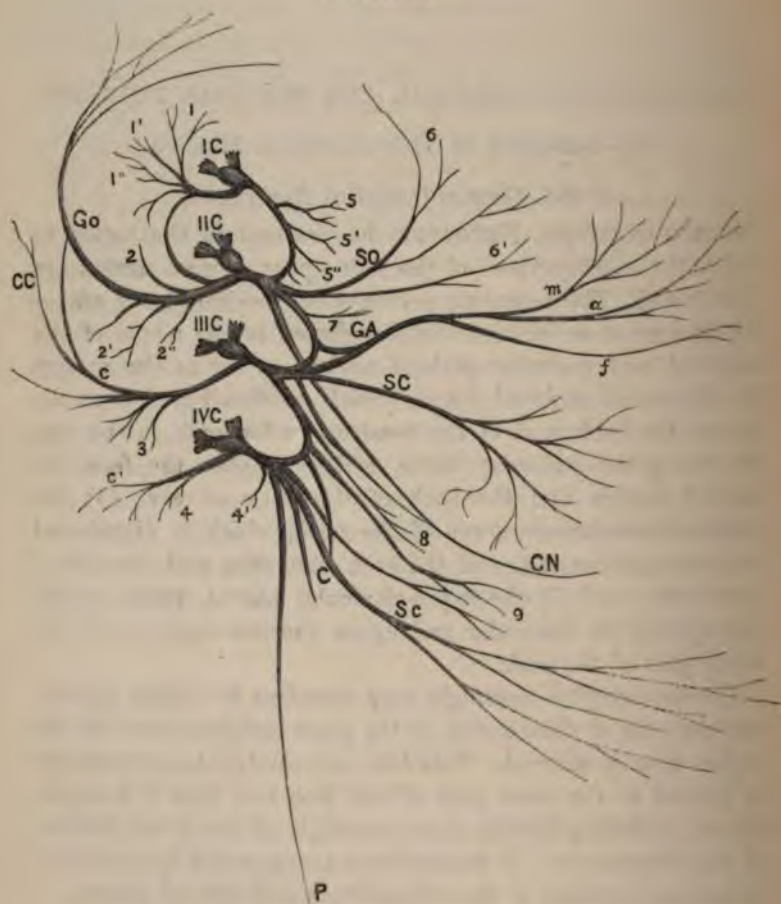
Cervico-occipital neuralgia may therefore be widely spread; but the area of distribution of the great occipital nerve is the region usually affected. This form of neuralgia has a tendency to spread to the lower part of the face, and then it becomes almost indistinguishable from neuralgia of the third division of the trigeminus. It is sometimes accompanied by great irritation and swelling of the submaxillary and cervical glands.

Cervico-occipital neuralgia is usually excited by exposure to draughts of cold air, and it may occasionally be the result of injury or disease of the upper cervical vertebræ.

*Painful points* are usually met with in this form of neuralgia; most frequently at the point of emergence of the great occipital nerve, about

midway between the mastoid process and the spinous processes of the upper cervical vertebrae (occipital point). The track of the nerve over the occiput is often painful. A point over the parietal eminence (the parietal point), and the spinous processes of the upper cervical vertebrae, are generally tender to the touch.

FIG. 59.

FIG. 59. *Nerves of the Cervical Plexus.*

IC, IIC, IIIC, IVC, First, second, third, and fourth cervical nerves.

- |      |          |  |
|------|----------|--|
| 1,   | Muscular | branch to rectus capitis posticus major and minor. |
| 1',  | "        | " obliquus superior.                               |
| 1'', | "        | " complexus.                                       |
| 2,   | "        | " obliquus inferior.                               |
| 2',  | "        | " complexus and trachelo-mastoid.                  |



The pain in this form of neuralgia usually radiates widely, most frequently affecting the brows, temples, and cheeks, which leads to its being sometimes mistaken for trigeminal neuralgia. The ordinary accompaniments of neuralgia may be observed, as hyperæsthesia of the skin of the occipital region, or anæsthesia, also spasm or tonic contractions of the cervical muscles.

The treatment of this form does not require any special mention, since it must be conducted on the general principles already detailed.

§ 287. *Phrenic Neuralgia*.—A form of neuralgia has been recently described by Peter,\* which appears to have its seat in the phrenic nerve. It is probable that the phrenic, instead of being, as has been supposed, purely motor, is a mixed nerve, although this idea yet awaits experimental confirmation. The symptoms are severe pain at the lower and anterior part of the thorax, along the line of attachment of the diaphragm, pain at the point where the nerve takes its origin from the cervical plexus and descends over the scalenus anticus, and also along the course of the nerve through the chest. Pain in the shoulder is also almost a constant and characteristic symptom of the affection.

- 
- 2", Muscular branch to splenius.
  - 3, " " muscles of the neck.
  - 4, " " muscles of the neck.
  - Go, Great occipital nerve, cutaneous to posterior part of scalp.
  - CC, Cutaneous branch to posterior part of scalp and back of neck.
  - 5, Muscular branches to rectus capitis lateralis.
  - 5', " rectus capitis anticus major and minor.
  - 5'', Communicating branches with hypoglossal and pneumogastric nerves.
  - SO, Small occipital nerve.
  - 6, Muscular branch to occipito frontalis muscle.
  - 6', Auricular branch to attollens aurem.
  - 7, Muscular branch to sterno-cleido-mastoideus, communicating with spinal accessory nerve.
  - GA, Great auricular nerve.
  - m, Mastoid branch to integument behind ear.
  - a, Auricular branch to external ear.
  - f, Facial branch to integument of face over parotid gland.
  - SC, Superficial cervical nerve to anterior and lateral parts of neck and muscular to platysma.
  - CN, Communicans noni joining descending branch of hypoglossal nerve.
  - 8, Muscular branch to levator anguli scapulae.
  - 9, " trapezius communicating with spinal accessory nerve.
  - Sc, Supra-clavicular nerves, or descending branches.
  - C, Communicating branch to brachial plexus.
  - P, Phrenic nerve.

\* Archiv. Général de Médecine, 1872, Tome xvii.

The painful points are (1) the spinous processes of the upper cervical vertebrae, especially from the second to the fifth; (2) the phrenic nerve itself in its course along the supra-clavicular fossa; (3) the line of attachment of the diaphragm, especially anteriorly between the seventh and tenth ribs, and more rarely posteriorly; (4) a point over the cartilage of the third rib, the significance of which is not obvious.

§ 288. *Spasm of the Muscles supplied by the Cervical and Dorsal Nerves.*

The following groups of muscular spasms may be distinguished in the region of distribution of the cervical and dorsal nerves.

1. *Spasm of Individual Muscles and Groups of Muscles in the Neck and Back, in the Shoulder, and in the Arm.*

(a) *Spasm of the Splenius Capitis.*—In spasm of this muscle the head is drawn backwards and towards the affected side; the chin somewhat depressed and directed towards the side of the spasm, and not to the opposite side, as in spasm of the trapezius; and a hard ridge can be felt at the point where the splenius appears beneath the anterior border of the trapezius.

FIG. 60.



FIG. 60. *Spasm of Splenius.*

The spasm is generally tonic, with remissions and occasional more energetic contractions.

(b) *Spasm of the Obliquus Capitis Inferior.*—Spasm of this muscle causes either intermittent or persistent rotation of the head around its vertical axis, without any elevation of the chin or depression of the mastoid process. In clonic spasm the patient may be observed often to correct the oblique position of the head with the hand when walking, or if he wishes to speak or to look at a fixed object.



(c) *Spasm of the deep muscles of the neck* is characterised by strong backward retraction of the head when the affection is bilateral, or towards the affected side when it is unilateral. A large proportion of all the cases of spasms in the neck, such as stiff neck and "boring of the head into the pillow," are due to implication of the muscles at the back of the neck.

The treatment is the same in principle as that for spasm of the sterno-mastoid.

2. *Spasms of the Respiratory Muscles, Inspiratory and Expiratory Cramps, Singultus, &c.*

(a) *Tonic Spasm of the Diaphragm.*—This affection is fortunately rare. It induces great dyspnœa, the patients being threatened with asphyxia. The lower half of the chest is expanded and immovable; the epigastrium strongly projects; whilst rapid and superficial respirations are performed with the upper part of the chest. There is severe pain in the epigastrium and along the attachments of the diaphragm, the patient is compelled to sit up in bed, the voice becomes feeble and muffled, and there is well-marked cyanosis; if the attack last beyond a short time, death ensues. This kind of spasm of the diaphragm is often the immediate cause of death in tetanus. It is occasionally a complication in tetany; but when it occurs as an unmixed affection, it is generally caused by exposure to cold, and on this account is usually regarded as a rheumatic affection of the diaphragm itself.

*Treatment.*—The treatment must be very energetic, as death may supervene in a few minutes unless relief is obtained. Inhalation of chloroform, subcutaneous injection of morphia, hot fomentations, faradisation with the brush and a strong current applied in the neighbourhood of the diaphragm, and the application of galvanic and faradic electricity in the course of the phrenic nerves, are the remedies to be mainly relied upon.

(b) *Clonic Spasm of the Diaphragm, Singultus, Hiccough.* Everyone is familiar with hiccough. It consists of short, energetic, spasmodic contractions of the diaphragm, accompanied by an inspiratory sound, which is usually suddenly arrested by the closure of the glottis. The contractions may succeed each other rapidly; often, indeed, so rapidly that a hundred

contractions occur in a minute. The attack may, on the one hand, last only a few minutes; and on the other, for hours, days, or weeks, and may recur more or less frequently for years.

When the spasms are violent severe pain is experienced in the epigastrium and along the attachments of the diaphragm. If the hiccough is frequent dyspnoea occurs, and the rhythm of the respiration and articulation is considerably disturbed. The ingestion of food is interfered with, digestion is imperfect, rest is disturbed, and the spasm is not always arrested during sleep. Spasm of the diaphragm may occasionally be caused by direct irritation of the phrenic nerve or of the respiratory centre. Hiccough has been observed in diseases of the central nervous system and in injuries of the skull and cervical portion of the spinal column. Amongst other causes of hiccough may be mentioned emotional disturbances, hysteria, malarial poison, chlorosis, and cachexia. Hiccough is, however, more frequently caused by reflex irritation, proceeding from the subjacent viscera. Simple repletion or pressure on the stomach may cause it, and it is frequently observed in gastric and intestinal diseases of all kinds, in peritonitis, in hepatic and uterine affections, in disturbances of menstruation, and in affections of the prostate gland. It may sometimes follow a prolonged fit of coughing, and has been observed associated with pericarditis. It is generally an ominous sign in cancer of the abdominal viscera and in other cachectic conditions.

The prognosis is in most cases favourable, although in hysterical and other nervous affections it often resists treatment in the most obstinate manner. The final hiccough in cachectic conditions naturally carries with it a bad prognosis.

*Treatment.*—The first object of treatment is to remove the cause. In slight cases mental impressions, such as fright and strong diversion of the attention in another direction, are often successful in removing the disease. A powerful expiratory effort sometimes removes the spasm; and Cruveilhier suggested a plan which he found successful, namely, to pour water into the mouth until the patient fears that he is about to be suffocated. These methods probably act in a reflex manner.

In order to induce the necessary expiratory effort the patient may be directed to hold his breath as long as possible, to strain



with closed glottis, or to inhale a strong odour or pungent smell so as to induce sneezing. When the spasm does not give way with these simple methods, hot fomentations or a blister may be applied over the diaphragmatic region. The faradic brush applied in full strength over the epigastrium and hypochondria is very effective. Galvanisation or faradisation along the course of the phrenic nerves is very useful.

Narcotics are also useful, the best being subcutaneous injection of morphia. Opium in other forms, cannabis indica, and atropine have also been successfully tried. The inhalation of ether or chloroform may be necessary in obstinate cases.

The nervine tonics, such as zinc, valerian, assafætida, arsenic, strychnine, and nitrate of silver, may be tried in severe cases.

(c) *Inspiratory Spasm (Spasmus Inspiratorius)*.—In inspiratory spasm there is a spasmodic rhythmic contraction of all, or almost all, the inspiratory muscles. True inspiratory spasm differs considerably from hiccough, although the latter frequently complicates the former. The essential feature of the affection is that either many or all of the muscles of inspiration participate in the spasm, and that a true inspiration, unbroken by sudden closure of the glottis, takes place. The spasm consists in a more or less rapid succession of deep inspirations, whilst the intervening expirations are performed in the usual noiseless way. The chest is powerfully expanded, the epigastrium is protruded, the auxiliary muscles of respiration are excited to action, the pectorales and sterno-cleido-mastoids are brought into strong relief, the shoulders are raised, the head is drawn backwards, and the respiratory muscles of the face, *alæ nasi*, and eyelids contract. Inspiration is noisy, and it is often accompanied by eructation of gas from compression of the stomach. The spasm usually occurs in paroxysms of variable duration, the abdomen is generally tympanitic, and there are, as a rule, other symptoms of nervous derangement, especially those characteristic of hysteria.

(d) *Attacks of Sneezing (Sternatatio Convulsiva)*.—Attacks of sneezing occur in a paroxysmal and spasmodic form, so that the patient will sometimes sneeze several hundred times in succession. Ordinary sneezing is a reflex act excited by irritation of the nasal filaments of the fifth pair. Attacks of sneezing are

generally accompanied by a profuse watery secretion from the nasal cavities; and when of long duration, they cause great misery to the patient.

(e) *Attacks of Yawning (Oscedo, Chasma).*—Chasma consists of a succession of yawns following each other with greater or less rapidity, and accompanied by the well-known phenomena of gaping, flow of saliva, secretion of tears, and hardness of hearing with dull tinnitus aurium.

(f) *Spasmodic cough* comprises all those paroxysmal attacks of coughing which are accompanied by a loud, ringing sound. Such attacks of coughing may last for a variable period, and may also recur frequently for months or years.

(g) *Fits of laughing or crying* are both forms of expiratory spasm. The former consists of a succession of loud expirations accompanied by vocal tones; and the latter consists of long-drawn expirations, often interrupted by sobs, accompanied by wailing or moaning sounds, and generally by a profuse secretion of tears. These actions are usually associated with well-known mental states, and accompanied by characteristic facial expressions; but they may be quite independent of emotional disturbances in pathological conditions, and are then designated as spasmodic. These spasms generally constitute subordinate symptoms of severe general neuroses, especially of hysteria, or are produced by disease of the central nervous system. These spasms may also be caused by reflex irritation in uterine diseases, pregnancy, intestinal worms, skin diseases, sexual excitement, hæmorrhoids, dysmenorrhœa; or in the case of sneezing from irritation of the mucous membrane of the nose, conjunctiva and disease of the ear.

*Treatment.*—The first object of treatment is to remove the cause, such as any source of reflex irritation or hysteria. The direct treatment of these forms of spasms must be conducted by cutaneous irritants, electro-therapeutics, narcotics, and anti-spasmodics. For the cure of sneezing, compression of the root of the nose, plunging the head into cold water, sponging the face and nose in hot water, irritations of the skin, emetics, inhalation of chloroform, and of the vapour of iodine, may be successively tried. Helmholtz has recommended the local



application of solution of quinine to the nasal mucous membrane in cases of hay asthma.

§ 289. *Paralysis of the Muscles of Inspiration.*

The muscles of inspiration are widely separated from one another in position, and are innervated by various nerves.

(a) *Complete paralysis* occurs when the respiratory centres in the medulla oblongata are affected. The medulla may be affected by various degenerative processes which implicate the respiratory centres, and the action of various poisons arrests their functional activity. Paralysis of the muscles of respiration may also occur when the motor tracts proceeding from these centres, which run in the lateral columns of the cord, are destroyed, as occurs in compression of the cord from fracture of the upper cervical vertebræ, and in these cases rapid death is inevitable.

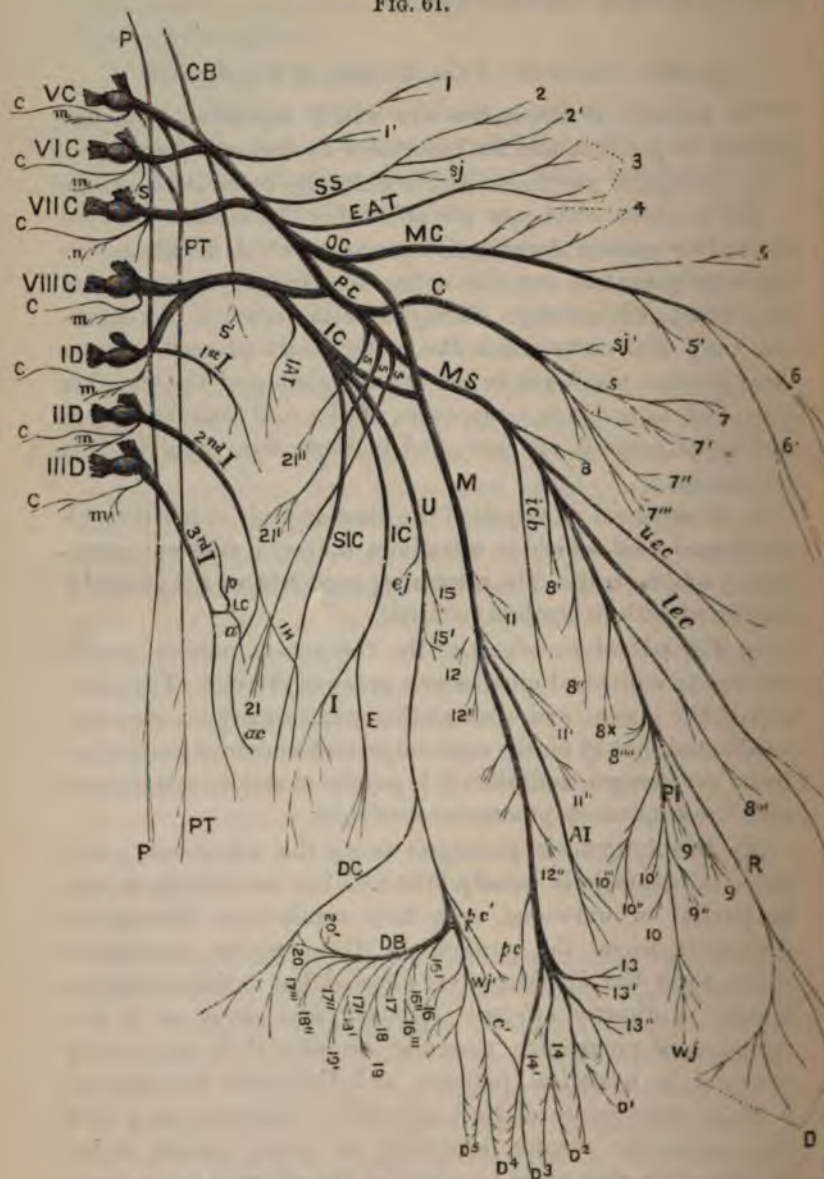
(b) *Unilateral paralysis* of the fibres in their course through the spinal cord, which is sometimes observed, does not immediately endanger life. The respiratory processes are not generally interfered with in cerebral paralysis.

(c) *Peripheral paralysis* of the respiratory muscles usually affect only individual muscles or a group of muscles. The intercostals, the scaleni, and other auxiliary inspiratory muscles may be affected; but by far the most important muscle of inspiration is the *diaphragm*, and when it is paralysed serious interference with the respiratory process is occasioned.

(d) *Diaphragmatic paralysis* is on the whole rare; and when it occurs it is usually bilateral, but occasionally it may be partial or unilateral. It may result from pleurisy or peritonitis when the portions of these serous membranes which cover the diaphragm are affected, but in these cases the muscle is directly affected. It may also occur as a late symptom of progressive muscular atrophy, it is occasionally observed in hysterical patients, and Duchenne has observed it as a consequence of lead poisoning. Exposure to a chill may cause the disease, especially in young people, either by producing rheumatic paralysis of the phrenic nerve or by causing muscular rheumatism of the diaphragm. Paralysis of

## (II).—DISEASES OF THE BRACHIAL PLEXUS.

FIG. 61.

FIG. 61 (after Flower). *Nerves of the Brachial Plexus.*

VC, VIC, VIIC, VIIIC, ID, IID, IIID, Fifth, sixth, seventh, and eighth cervical and first, second, and third dorsal nerves.  
 CB, Communicating branch from the fourth cervical nerve.



- P, Phrenic nerve.  
 c, c, Dorsal cutaneous branches from the fifth cervical to the third dorsal nerve.  
 M, M, Branches to the muscles of the neck and back.  
 B, Branch to the scalenus medius.  
 S', Branch to the subclavius muscle.  
 I, I', Branches to Rhomboideus major and Rhomboideus minor.  
 SS, Supra scapular nerves :—  
     2, Branch to supra spinatus muscle.  
     2', " infra spinatus muscle.  
     sj, " shoulder joint.  
 PT, Posterior or long thoracic (external respiratory of Bell) supplies serratus magnus.  
 EAT, External anterior thoracic supplies pectoralis major.  
 IAT, Internal anterior thoracic to pectoralis major and pectoralis minor.  
 1I, First intercostal nerve.  
 2I, Second intercostal nerve.  
     IH, Intercoasto-humeral joins nerve of Wrisberg.  
     AC, Anterior cutaneous nerves of the thorax.  
 3I, Third intercostal nerve.  
     LC, Lateral cutaneous :—  
         a, Anterior branch.  
         p, Posterior branch.  
 OC, Outer cord of brachial plexus.  
 PC, Posterior cord " "  
 IC, Inner cord.  
 MC, Musculo-cutaneous nerve.  
     4, Branches to coraco-brachialis.  
     5, " biceps.  
     5', " brachialis anticus.  
     6, Anterior cutaneous branch } to outer side of forearm.  
     6', Posterior cutaneous branch }  
 IC', Internal cutaneous nerve.  
     E, Anterior or external branch.  
     I, Posterior or internal branch to inner side of forearm.  
 SIC, Small cutaneous nerve (nerve of Wrisberg to inner side of arm.  
 SSS, Subscapular nerves :—  
     2I, Long subscapular nerve to latissimus dorsi.  
     2I', Muscular branches to subscapularis and teres major.  
     2I'', " " subscapularis.  
 ;, *Circumflex Nerve.*  
     sj, Branch to shoulder joint.  
     s, Superior division.  
         7, Cutaneous.  
         7', Muscular to deltoid.  
     i, Inferior division.  
         7'', Cutaneous.  
         7'', Muscular to teres minor.  
 IS, *Musculo-spiral Nerve.*  
     8, Muscular to brachialis anticus.  
     8', " triceps.  
     8'', " anconeus.  
     8''', " supinator longus.  
     8''', " supinator brevis.  
     8x, " extensor carpi radialis longior.  
     icb, Internal cutaneous branch to inner side of arm.  
     uec, Upper external cutaneous branch to outer side of arm.  
     lec, Lower external cutaneous branch to outer side and back of forearm.  
 R, Radial nerve cutaneous to dorsal surface of thumb and two outer fingers.  
 PI, Posterior Interosseous.  
     9, Muscular branch to extensor carpi radialis brevior.  
     9', " " " ossis metacarpi pollicis.  
     9'', " " " primi internodii pollicis.  
     10, " " " secundii internodii pollicis.  
     10', " " " indicis.  
     10'', " " " carpi ulnaris.  
     10''', " " " digitorum communis and extensor minimi digiti.  
     wj, Branch to wrist joint.

the phrenic nerve may also be caused by wounds or the presence of tumours in the neck, and under these circumstances the paralysis is apt to be unilateral.

The symptoms of diaphragmatic paralysis are highly characteristic. During inspiration the epigastrium and hypochondria are drawn inwards instead of being curved outwards, and if the hand be placed on the epigastrium during inspiration, the pressure of the descending diaphragm cannot be perceived; while, on the other hand, the epigastrium projects on expiration. When the paralysis is unilateral, these symptoms occur only on one side, but they may be distinctly detected by palpation.

During rest the frequency of inspiration is not much increased, but if the slightest exertion is made dyspnoea is at once experienced, and the frequency of respiration rises to 40 or 50 in a minute. The patient is in great danger, and if an

**M, Median Nerve.**

- 11, Muscular branches to pronator radii teres.
- 11', " " flexor profundus digitorum.
- 11'', " " flexor longus pollicis.
- A1, Anterior interosseous branch to pronator quadratus.
- 12, Muscular branch to flexor carpi radialis.
- 12', " " flexor sublimis digitorum.
- 12'', " " palmaris longus.
- 13, " " opponens pollicis.
- 13', " " abductor pollicis.
- 13'', " " flexor brevis pollicis (outer half).
- 14, " " first lumbricalis.
- 14', " " second lumbricalis.
- pc, Palmar cutaneous branch.
- D to D4, Digital cutaneous branches.

**U, Ulnar Nerve.**

- ej, Branch to elbow joint.
- 15, Muscular branch to flexor profundus digitorum (inner part).
- 15', " " carpi ulnaris.
- DC, Dorsal cutaneous branch. To dorsal surface of two inner fingers.
- p'e, Palmar cutaneous branch.
- e', communicating to median.
- D3 to D4, Cutaneous to little finger and inner side of ring finger.
- 16, Muscular branches to palmaris brevis.
- 16', " " abductor minimi digiti.
- 16'', " " opponens minimi digiti.
- 16''', " " flexor brevis minimi digiti.
- 17, " " fourth dorsal interosseous.
- 17', " " third " "
- 17'', " " second " "
- 17''', " " first " "
- 18, " " third palmar interosseous.
- 18', " " second " "
- 18'', " " first " "
- 19, " " fourth lumbricalis.
- 19', " " third " "
- 20, " " adductor pollicis.
- 20', " " flexor brevis pollicis (inner half).



intercurrent attack of bronchitis or pneumonia supervene, the danger is greatly increased, inasmuch as the power of inspiration is diminished. The compressive action of the abdominal muscles is materially interfered with in diaphragmatic paralysis, inasmuch as no counter pressure is obtainable, hence considerable difficulty in defecation is experienced.

The diagnosis of diaphragmatic paralysis presents no special difficulty. The prognosis depends upon the cause. It is favourable in rheumatic and hysterical paralysis; it is doubtful in lead paralysis, and very unfavourable in progressive muscular atrophy, although even in the latter case considerable improvement may be produced by appropriate treatment.

*Treatment.*—The treatment must first be directed to remove the cause. Recourse should be had at an early period to faradisation or galvanisation of the phrenics, which can be easily applied in the neck over the scaleni. Irritation is best effected by placing one pole upon the neck whilst the other is placed in the vicinity of the attachments of the diaphragm to the ribs, or, in some instances, to the back of the neck, and the current should be tolerably strong.

### § 290. *Cervico-Brachial Neuralgia.*

*General Characters of the Disease.*—This group includes all the neuralgias which occur in nerves originating from the brachial plexus, or from the posterior branches of the four lower cervical nerves. The pain is seldom limited to one branch of the nerve, the disease, as a rule, involving several. The pain may be seated in the upper arm or forearm, or it may extend into the hands and fingers. Owing to the intimate interweaving of the various nerve trunks in the brachial plexus, it is not easy to determine what branches of the plexus, or what special nerve roots, are implicated. It is only when the neuralgia affects some part near the periphery that the pain is limited to any particular branch of the plexus.

*Etiology.*—The predisposing causes of cervico-brachial are the same as those of cervico-occipital neuralgia; but hysteria and anæmia appear to be specially frequent causes of this form of neuralgia, hence females are more frequently affected by it than males.

The most important of the exciting causes of the disease are the injuries of all kinds to which the upper extremities are so peculiarly exposed. Exposure to cold and over-exertion are also frequent exciting causes of the disease. Mr. James Salter has shown that cervico-brachial neuralgia may occasionally originate from reflex irritation set up by carious teeth. Lead poisoning and malaria produce this form of neuralgia, and it may be a symptom of central disease, as tabes, hemiplegia, and progressive muscular atrophy.

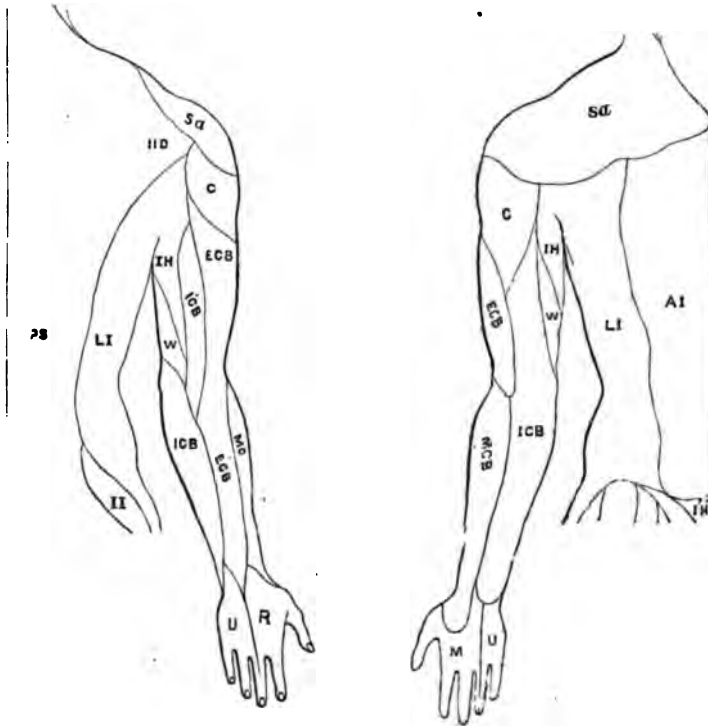
*Symptoms.*—The pain of cervico-brachial neuralgia is of a constant, dull, boring, burning character, and is very severe. Lancinating pains are also experienced, which shoot through the arm; sometimes in the ascending, sometimes in the descending direction, and generally following the course of the principal nerve trunks. The violent burning described by Weir Mitchell, under the name of "Causalgia," is often present in the neuralgias which are caused by gunshot injuries of the nerves. The pain often occurs in *nocturnal paroxysms*, which last through the night, and may almost disappear during the day. A paroxysm may be revived or aggravated by movements of the muscles of the arm, such as those involved in playing the piano, sewing, or other manual exertions.

*Painful Points.*—Owing to the numerous peripheral anastomoses of the branches of the nerves, the painful points are somewhat indefinite. The following have been distinguished by Valleix, and confirmed by Dr. Anstie: 1, *An axillary point*, corresponding to the brachial plexus itself; 2, a *scapular point*, corresponding to the lower angle of the scapula, which is difficult to explain; 3, a *shoulder point*, which corresponds to the emergence, through the deltoid muscle, of the cutaneous branches of the circumflex; 4, a *median-cephalic point*, at the bend of the elbow, where a branch of the musculo-cutaneous nerve lies immediately behind the median-cephalic vein; 5, an *external humeral point*, about three inches above the elbow, corresponding to the emergence of the cutaneous branches which the musculo-spiral gives off as it lies in the groove of the humerus; 6, a *superior ulnar point*, corresponding to the course of the ulnar nerve between the olecranon and the epitrochlea; 7, an *inferior ulnar point*, where the ulnar nerve passes in front of the annular ligament of the wrist; 8, a *radial point*, marking the place where the radial nerve becomes superficial at the lower and external aspect of the forearm. Painful points may occasionally be developed by the side of the lower cervical vertebrae, corresponding to the posterior branches of the lower cervical nerves.



*Concomitant* symptoms are almost always present in every case of cervico-brachial neuralgia. In some cases a certain amount of anæsthesia is present, associated with numbness and

FIG. 62.

FIG. 62 (after Flower). *Cutaneous Nerves of the Trunk, Upper Extremity.*

Sa, Supra clavicular nerve.

IID, Second dorsal.

PS, Posterior branches of the spinal nerves.

LI, Lateral branches of the intercostal nerves.

AI, Anterior branches of the intercostal nerves.

II, Iliac branch of ilio-inguinal nerve.

I'H, Ilio hypogastric nerve.

C, Circumflex nerve.

IH, Intercosto humeral.

W, Nerve of Wrisberg.

ICB, Internal cutaneous branch of musculo-spiral nerve.

ECB, External cutaneous branch of musculo-spiral nerve.

ICB, Internal cutaneous nerve.

MC, Musculo cutaneous.

R, Radial nerve.

U, Ulnar nerve.

M, Median nerve.

formication, while in other cases hyperæsthesia exists in the affected region of the skin to such an extent as to render contact with the bed-clothes unendurable. Radiating pains are felt in the region of distribution of the cervical plexus and of the upper dorsal and intercostal nerves.

The motor power of the affected extremity is impaired in almost all cases. The movements of the arm are rendered difficult and painful, the arm and fingers are stiff, and twitchings and fibrillary contractions are not uncommon, and occasionally there may be persistent spasmodic contractions, or paresis, or even paralysis of particular muscles, or groups of muscles.

*The vaso-motor and trophic disturbances* may become manifest in some cases in the form of coldness and pallor, while in other cases the reverse conditions, heat and redness, obtain. A plentiful crop of *Herpes* is not an uncommon symptom, and the more severe trophic disturbances declare themselves in the various forms of pemphigus, obstinate ulcers, glossy skin, and changes in the growth of the nails and the like. The latter changes are induced by severe injuries to nerves, and they are always accompanied by serious motor and sensory paralysis. In cervico-brachial neuralgia the state of the heart and large vessels ought to be carefully ascertained.

*Diagnosis.*—Cervico-brachial neuralgia may be mistaken for many kinds of painful affections, as muscular and articular rheumatism, diseases of bone, &c., and this mistake can only be avoided by a careful examination into all the circumstances of the case.

*Treatment.*—The treatment ought first to be directed to the removal of the cause of the disease; and, considering how frequently this form of neuralgia is caused by external injuries and local growths, great scope is afforded for surgical interference.

Electricity is the most useful agent for the treatment of the neuralgia as a symptom. The constant current is most generally useful, although the faradic current sometimes succeeds when the other fails.

Narcotics are both useful and necessary in the treatment of severe cases. Subcutaneous injection of morphia is the most generally useful; but sometimes great benefit is obtained from the use of belladonna or atropine. External applications are also useful. The most generally employed are liniments of



chloroform, and ointments containing opium, and especially veratria. The various forms of counter-irritation may be sometimes employed, and Weir Mitchell recommends repeated vesication in the treatment of "causalgia." For internal treatment, quinine, iron, and arsenic are the remedies to be mainly relied upon. Cold or hot water treatment, in the form of baths, shampooing, and fomentations, may be employed according as the one or other is found to afford the greatest amount of relief.

Neurectomy of mixed nerves should only be resorted to under pressing necessity, and only when distinct evidence has been obtained of the existence of a peripheral cause for the neuralgia. And what is true of neurectomy in this respect is applicable to a still greater extent with regard to amputation or resection as a means of obtaining relief.

§ 291. *Spasm of Special Muscles and Groups of Muscles of the Neck and Trunk.*

(1) *Spasm of the Rhomboidei* manifests itself by the peculiar position of the scapula. The lower angle is raised so that the inner border assumes an oblique position from above downwards, and from without inwards; whilst it is at the same time approximated to the vertebral column. The muscle can be felt as a firm swelling between the spinal column and scapula. This affection is distinguished from paralysis of the serratus magnus by the fact that the internal border of the scapula is not lifted away from the chest, and the shoulder is not depressed. A distinct resistance is experienced when an attempt is made to raise the scapula to a vertical position,

FIG. 63.



FIG. 63 (Duchenne). *Contraction of Rhomboid Muscles.*—A, Levator scapulae; B, Retracted rhomboid; C, Fibres of serratus magnus; D, Abnormal position of the inferior angle of the scapula; E, Inferior angle on the healthy side.

on account of the antagonism of the rhomboid. Tonic spasm or contracture of this muscle has hitherto alone been observed.

(2) *Spasm of the Levator Anguli Scapulæ*.—The scapula is strongly elevated in this affection, especially its upper and inner angle; the head is slightly inclined to the same side; the shoulder is drawn somewhat forwards; the supra-clavicular fossa is increased in depth, and the muscle it contains projects distinctly beneath the anterior border of the trapezius, which can easily be isolated from it by faradisation. It frequently occurs in the form of a tonic contracture in combination with spasm of the rhomboidei or of the trapezius.

(3) *Spasm of the serratus magnus, of the latissimus dorsi, of the teretes, of the supra and infraspinati, of the subscapularis, and of the pectoralis major* may generally be readily recognised by the characteristic positions and movements of the scapula and arm (elevation, depression, rotation, adduction, and abduction) in combination with distinct hardness and fulness in the regions of the several muscles affected. Spasms of these muscles are rare.

#### § 292. *Spasm of the Muscles of the Upper Extremity.*

(1) *Spasm of the Muscles supplied by the Circumflex Nerve*. The circumflex nerve supplies the deltoid and teres minor muscles, and gives branches to the skin of the upper arm and shoulder joint. In the tonic form of this affection the arm is held out from the body, and is directed backwards in cases where the posterior fibres of the deltoid and the teres minor are affected. The lower angle of the scapula is pressed backwards towards the vertebral column, as in paralysis of the serratus magnus. In cases of clonic spasm of the deltoid, the arm is thrown upwards and moved convulsively in various directions; and other muscles are usually implicated.

(2) *Spasm of the Muscles supplied by the Musculo-Cutaneous Nerve*.—The muscles supplied by the musculo-cutaneous nerve are the coraco-brachialis, the biceps, and the brachialis anticus; but the last receives additional branches from the musculo-spiral nerve. The musculo-cutaneous nerve passes through the



coraco-brachialis muscle, and is then found in the interval between that muscle and the biceps, or further outwards between the two heads of the biceps. Spasm of the muscles supplied by this nerve causes strong flexion of the forearm.

(3) *Spasm of the Muscles supplied by the Musculo-Spiral Nerve.*—The musculo-spiral nerve supplies the triceps, anconeus, a small part of the brachialis anticus, and all the extensor and supinator muscles of the forearm. When the muscles supplied by the musculo-spiral nerve are in a state of spasm, the forearm is supinated, the hand and thumb completely extended, while there is also extension of the first phalanges of the fingers.

(4) *Spasm of the Muscles supplied by the Median Nerve.*—The median nerve supplies the pronators, the flexor carpi radialis, the flexor digitorum sublimis, the radial half of the flexor profundus digitorum, the flexor longus pollicis, two outer lumbricales, and the small muscles of the ball of the thumb, with the exception of the adductor and the ulnar head of the flexor brevis pollicis. When the muscles supplied by the median nerve are in a state of spasm the forearm is strongly pronated, the hand is bent towards the radial side, and the fingers flexed with opposition of the thumb.

FIG. 64.

FIG. 64 *Muscles of the Hand* (from Wilson).

- 1, Annular ligament.
- 2, 2, Origin and insertion of the abductor pollicis muscle.
- 3, Opponens pollicis.
- 4, Superficial portion of the flexor brevis pollicis.
- 5, Deep portion of the flexor brevis pollicis.
- 6, Adductor pollicis.
- 7, 7, The lumbricales muscles, arising from the deep flexor tendons, upon which the figures are placed. The tendons of the flexor sublimis have been removed.
- 8, Insertion of one of the tendons of the deep flexor.
- 9, The tendon of the flexor longus pollicis, passing between the two portions of the flexor brevis to the last phalanx.
- 10, Abductor minimi digiti.
- 11, Flexor brevis minimi digiti.
- 12, Pisiform bone.
- 13, First dorsal interosseous muscle, the abductor indicis.

Spasm of the muscles of the hand supplied by the median produces opposition of the thumb, with approximation and slight flexion of the first phalanges of the index and middle fingers.

(5) *Spasm of the Muscles supplied by the Ulnar Nerve.*—The ulnar nerve innervates the flexor carpi ulnaris, the ulnar half of the flexor profundus digitorum, all the muscles of the

FIG. 65.

FIG. 65. *Attachment of an Interosseous Muscle* (from Duchenne).

- a, Interosseous muscle.
- b, Attachment to base of first phalanx.
- c, Slip passing forward to (d) side of extensor tendon.
- e, Central portion of extensor tendon.

hypothenar eminence, together with the palmaris brevis, all the interossei, the two ulnar lumbricales, the adductor pollicis, and the inner head of the flexor brevis pollicis. The actions of the interossei in the movements of the fingers have been carefully investigated by Duchenne, and are so important as to deserve special mention. The three palmar interossei are arranged as adductors to and the four dorsal as abductors of the

FIG. 66.

FIG. 66. *Insertion of Muscles of Thumb* (from Duchenne).

- a, Abductor pollicis.
- b, Opponens pollicis.
- c, Outer head of flexor brevis.
- d, Tendon of extensor secundi internodii.
- e, Tendinous expansion of flexor brevis joining tendon of extensor.



fingers from an imaginary line drawn through the long or middle finger. The insertion of the interossei into the base of the first phalanx enables them to act as flexors at the metacarpophalangeal joint, whilst giving lateral movements to the fingers to which they are attached. The slip (*Fig. 65, d*) sent forward to join the extensor tendons extends the second and third phalanges. The interossei, therefore, produce flexion at the first and extension of the second and third phalanges, and also assist both to adduct and abduct the fingers in relation to the middle line of the hand. The lumbricales act with the interossei as flexors of the first, and extensors of the second and third phalanges. The small muscles of the thumb act in a similar manner. The two heads of the flexor brevis pollicis are inserted into the sides of the base of the first phalanx of the thumb, sending slips forward to join the tendons of the extensor secundi interodii pollicis on the back of the first phalanx (*Fig. 66, e*). This arrangement enables the small muscles of the thumb to extend the second phalanx, whilst acting on the first in the direction implied by their several names. In spasm of the muscles supplied by the ulnar nerve the hand is rendered concave, the thumb adducted, the little finger strongly flexed and opposed, and the remaining fingers moderately flexed at the metacarpophalangeal, and extended at the phalangeal articulations. The position assumed by the hand in spasm of the interossei is represented in *Fig. 67*.



FIG. 67 (after Gowers). Position of the Hand in Spasm of the Interosseous Muscles.

(6) *Spasms of the muscles of the arms* occur in various modes and combinations. If the electrode of a faradic current be placed at the external edge of the sterno-mastoid muscle on a level with the transverse process of the sixth cervical vertebra, the deltoid, biceps, coraco-brachialis, the long and short supinator muscles will enter into contraction. These muscles are supplied by the musculo-cutaneous, circumflex, and musculo-spiral nerves; but it is probable that the fibres which supply the muscles are all found in the cord formed by the fifth and sixth cervical nerves, as it lies between the scalenus medius and posticus muscles. This is an important

point, inasmuch as combined paralysis of these muscles is not unfrequently observed. There are other cases in which more or less spasmodic irritation has been observed to affect the greater number of the muscles of the upper extremity, as in the case of "ascending contracture" described by Duchenne, which often spreads over the greater part of the upper extremities in consequence of irritation of the joints. Alcoholic tremor and that of paralysis agitans are sometimes limited to the upper extremities. Unilateral epilepsy is not unfrequently ushered in by a convulsive aura of one arm. The spasmodic movements which have been described under the names of athetosis and post hemiplegic chorea, and which are usually limited to the hand and forearm, will be subsequently described. Weir Mitchell has described spasmodic movements in stumps left after amputation of the arm; they caused constant movement of the stump, and were regarded by him as of reflex origin.

The causes of spasm of the muscles of the arm may be peripheral or central, and it may be due to rheumatism or to some kind of reflex action; and in some cases spasms occur in the absence of any recognisable cause. The diagnosis and prognosis must be based on general principles and on a thorough examination of the patient, and upon the seat, causes, and duration of the disease.

*Treatment.*—The treatment of spasm of these muscles is the same as for spasms of muscles in general. Duchenne recommends in cases of contracture faradisation of the antagonist muscles, and cutaneous faradisation in rheumatic cases of recent origin.

§ 293. *Writers' Cramp* — *Graphospasmus* — *Mogigraphia* (Pianists' Cramp, Tailors' Cramp, Milkers' Cramp, &c.).—Writers' cramp is only one of a large group of affections which have been called *professional hyperkineses*. This name has been given to these affections because the spasmodic movements always affect muscles engaged in delicate associated and acquired actions, such as those engaged in writing, pianoforte playing, sewing, &c.

Benedict describes three varieties of writers' cramp, namely, the spastic, the tremulous, and the paralytic form.



*Symptoms.*—The disturbances of movement are at first slight, and may only amount to a sensation of great weariness when the act of writing is long continued. After a time the symptoms become more marked, and make their appearance soon after the patient begins to write, or even immediately the pen is taken in the hand.

(1) In the *spastic* form of the disease, tonic or clonic spasms of one or of several of the muscles occur. These spasms are at first limited to particular fingers, causing an irregular stroke in the writing. The patient cannot write while holding his pen in the usual position, but he can do so tolerably well by holding it in a new and more or less grotesque manner (Poore). After a time the spasms become stronger, and are generally tonic in character, affecting usually the thumb and first finger. The thumb and first finger may be suddenly extended, causing the pen to drop; or there is spasmodic action of the *opponens pollicis*, with abduction and flexion of the index finger, so that the pen is rapidly moved away from the paper. At other times there is a spasmodic flexion of the first three fingers, so that they are pressed tightly against the pen, which cannot then be moved further onwards; or there may be movements of pronation and supination in the forearm, so that the pen is raised from the paper, and moved backwards and forwards in the most irregular manner. The faradic contractility of the affected muscles is sometimes increased, and at other times diminished, the former probably indicating an early, and the latter a late stage of the affection (Poore).

(2) In the *tremulous* form of the disease, the hand and forearm, or even the whole arm, become the subjects of well-marked tremors on any attempt at writing being made, so that the pen only makes undulating or angular strokes, and the writing becomes completely illegible.

The patient adopts many expedients to prevent the occurrence or to counteract the effects of the spasms; but, in spite of all, the handwriting, when writing is possible, becomes completely altered in character. The strokes are coarse, imperfect, and unequal, and numerous irregularities and false strokes are to be observed; and in the highest degrees of the affection the writing becomes a mass of undulating and zigzag strokes, and wholly illegible.

(3) The *paralytic form* offers a great contrast to the spastic and tremulous forms. In this form great fatigue and weakness of the hand and forearm are experienced when the patient attempts to write. As soon as the pen is laid down the feeling of weakness and exhaustion disappears, to reappear as soon as it is taken up again. It is generally confined to the flexors, or to the extensors muscles.

Those who suffer from writers' cramp also suffer from impairment of the movements requisite for sewing, pianoforte playing, embroidery, buttoning-up the clothes, and all actions requiring delicate manipulation; and, if the patient has learned to write with his left hand, the spasm frequently, to his great disappointment, extends to this also.

The most common *sensory disturbance* is the painful feeling of fatigue in the affected extremity, which may rise to a high degree of intensity and assume a neuralgic character. The pain frequently extends to the shoulder and back, and some of the spinous processes of the cervical and dorsal vertebræ are not unfrequently sensitive to pressure. Formication and numbness are frequently complained of. True anæsthesia is rarely present, and the same may be said of hyperæsthesia, and it is only very occasionally that the presence of pressure points has been ascertained; and in these cases it is probable that neuritis has been present.

Other spasmodic disturbances are not unfrequently associated with writers' cramp. Amongst the most frequent of them may be mentioned strabismus, stammering, spasm of the face, throat, and other parts of the body; weakness of the lower extremities with tremors occasionally occur, and as a rule there is great mental irritability and depression. The reaction of the affected muscles to electricity is normal, or only subject to a slight deviation, in the way of an excess or diminution of the electric contractility.

A few of the other professional hyperkineses may be briefly mentioned.

*Pianoforte Players' Spasm* is not of uncommon occurrence in professional players, especially women. It presents the same features as writers' spasm, and requires no separate description.

*Violin Players' Spasm* sometimes occurs in the left, sometimes in the



right hand, either in the form of painful exhaustion and stiffness, or as convulsive spasm of some of the muscles of the head, arm, or shoulder. It renders playing impossible.

*Tailors' and Shoemakers' Spasms* are somewhat analogous, and as soon as the patient begins to work, tonic or clonic spasms or functional debility of the muscles of the hand and arm are experienced.

The number of these forms of spasms might be largely increased. Indeed they may occur in any avocation requiring the constant associated action of certain groups of muscles. Such spasmodic muscular movements have already been observed in smiths, milkers, painters, makers of artificial flowers, harp-players, watchmakers, and turners.

*The Course* of all these diseases is almost always the same. They begin very gradually, and after a time increase more rapidly, and it is only in rare instances that they are observed to begin almost suddenly—after some powerful exciting cause, as over-exertion.

The *duration* of the disease is generally very protracted, often lasting through life. Its progress may often be arrested, but complete recovery is rare.

*Etiology.*—Writers' spasm is met with most frequently in men. The reason of the comparative immunity of women is probably that they are not so often called upon to over-exert themselves in writing as men. Pianoforte players' spasm, on the other hand, occurs more frequently in women. Writers' cramp is often inherited, or several members of a family may become affected by it. The main cause of the disease is excessive writing, hence it is most frequently observed in writers, secretaries, clerks, and merchants.

The spasm may occasionally be caused by exposure to cold, injuries to nerves or muscles, foreign bodies in the fingers, or from reflex action consequent on periostitis of the external condyle of the humerus. The spasms may sometimes be caused by centric disease or neuritis of one or other of the nerve trunks of the forearm; but such cases do not strictly belong to true writers' spasm.

The pathology of writers' cramp is somewhat obscure, since pathological anatomy has not hitherto thrown any light upon the question. Some writers believe that there is debility or paralysis of certain muscles and secondary spasm of their antagonists; but this supposition will not account for the facts. Fritz thought that in writers' cramp there is reflex spasm proceeding from the sensory cutaneous, or sensory muscular nerves;

but this hypothesis does not appear to be adequate for the explanation of the majority of cases. The fact that the movements which are acquired late in life under the guidance of the will are specially affected would appear to indicate that all forms of professional spasm are caused by a central lesion.

The *diagnosis* of writers' cramp presents no special difficulty. The diseases with which it is most likely to be confounded are the various forms of tremor, chorea, paralysis agitans, progressive muscular atrophy, locomotor ataxia, arthritis deformans, hemiplegia with contracture, and disease of the motor nerves of the hand, more especially of the ulnar (Poore); but a careful examination of each individual case should prevent anyone from falling into the error of mistaking writers' cramp for one of these affections. In order to determine with precision the nature of the disturbance and the particular muscles which are affected, the character of the handwriting must be studied, and each muscle carefully tested. Search should also be made for a peripheral source of irritation, such as the use of bad writing materials, the presence of a painful scar or other reflex irritation, or an attack of neuritis.

The *prognosis* is unfavourable. Arrest or even considerable improvement of the disease is not uncommon; but complete cure is rare. In a large number of cases the disease makes steady progress, and at length renders writing impossible in spite of every treatment. Writers' spasm has no direct influence on the general health, nor on the duration of life.

*Treatment.*—Removal of the cause is the primary requirement. Writing, pianoforte playing, or whatever else may have induced the disease, must either be entirely discontinued or limited as much as possible. In recent and slight cases this alone will effect a cure in from one to two months; but in severe cases there is little chance of recovery unless the patient can give up his occupation for six months or a year. If this cannot be done, the patient must be recommended to use soft pens, to write slowly and deliberately, and to use a suitable penholder. Erb has found thick cork penholders very serviceable.

Electricity is the most powerful remedy for the disease which



we possess. The *faradic* current appears to have little effect, but may prove useful in cases where there is local anæsthesia or hyperæsthesia, or paralysis of particular muscles. The spastic forms are made worse by the faradic current.

The *galvanic* current, on the other hand, frequently gives favourable results. Different methods of applying the current have been advocated by various electro-therapeutists; but the best known method appears to be to galvanise the vertebral column in the cervical region with ascending stabile or labile currents, and to combine with this the peripheral galvanisation of the nerves and muscles of the arm specially affected. Erb recommends also the transmission of galvanic currents, both transversely and in an antero-posterior direction through the head. The galvanic treatment should be continued several months at the least, and the application may be made as often as from three to six times per week; but the current employed must not be too strong.

When electric treatment proves ineffective, not much benefit need be expected from other remedies; but several other means may be employed along with electricity. The most promising of these methods are gymnastics, shampooing, tonics, mountain travelling, and a moderate cold-water cure. Baths, counter-irritants, and embrocations are of no avail. I have found benefit accrue in the paralytic form of the disease from subcutaneous injections of strychnia.

*Mechanical means* have been resorted to in desperate cases. The simplest method of this kind is to insert the pen into a cork or thick piece of wood, or to fasten it by means of a ring to the first or middle finger. The attempts which have been made to counteract particular spasmodic movements by means of complicated apparatus have all proved unsuccessful. Many patients are relieved by applying a narrow bandage or a strip of court plaster firmly round the wrist.

*Tenotomy* of the affected muscles has been performed by Stromeyer, Dieffenbach, Langenback, and others, but the results obtained have not been very encouraging. The treatment of the other *professional spasms* must be conducted on essentially similar principles.

§ 294. *Paralysis of Special Muscles and Groups of Muscles of the Neck and Trunk.*

(1) *Paralysis of the Pectoralis Major and Pectoralis Minor.*  
These muscles are supplied by the anterior thoracic nerves, and

FIG. 68.

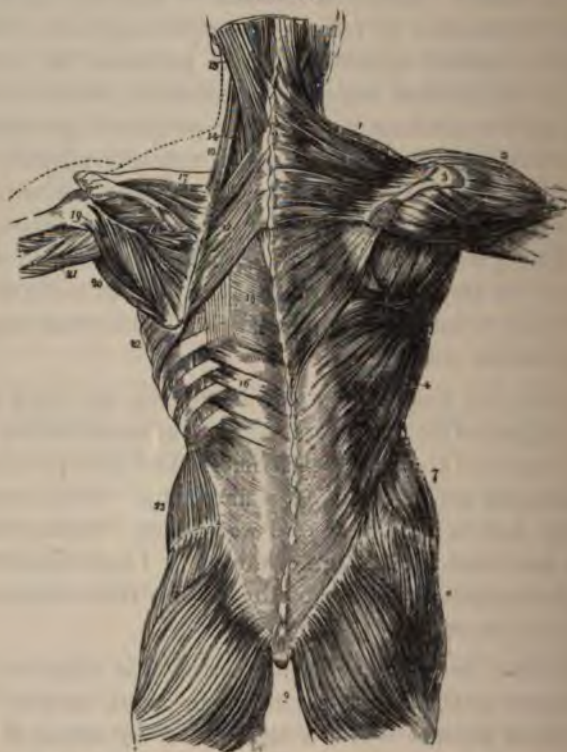


FIG. 68 (From Heath's "Anatomy"). First, second, and part of the third layer of muscles of the back.—The first layer occupies the right; the second the left side.

- |                                       |   |
|---------------------------------------|---|
| 1, Trapezius.                         | teral to, and overlaid by, the          |
| 2, Ligamentum nuchæ.                  | splenius, is the complexus.             |
| 3, Acromion process and spine of the  | 14, Splenius colli, partially seen; the |
| scapula.                              | common origin of the splenius is        |
| 4, Latissimus dorsi.                  | seen attached to the spinous pro-       |
| 5, Deltoid.                           | cesses below the origin of rhom-        |
| 6, Muscles of the dorsum of the right | boideus major.                          |
| scapula: infraspinatus, teres         | 15, Vertebral aponeurosis.              |
| minor, and teres major.               | 16, Serratus posticus inferior.         |
| 7, Obliquus externus.                 | 17, Supraspinatus.                      |
| 8, Gluteus medius.                    | 18, Infraspinatus.                      |
| 9, Glutei maximi.                     | 19, Teres minor.                        |
| 10, Levator anguli scapulae.          | 20, Teres major.                        |
| 11, Rhomboideus minor.                | 21, Long head of triceps.               |
| 12, Rhomboideus major.                | 22, Serratus magnus.                    |
| 13, Splenius capitis; the muscle in-  | 23, Obliquus internus.                  |



are rarely separately paralysed. There is impairment or loss of the power of adducting the arm to the thorax, and the patient is unable to seize the opposite shoulder with the hand, or to resist passive abduction of the arm. If the muscles are also atrophied, the sub-clavicular fossa is considerably deepened, the ribs and intercostal spaces are strongly marked, and the anterior wall of the axilla is reduced to a flaccid fold of skin.

(2) *Paralysis of the Rhomboidei and Levator Anguli Scapulæ.* All these muscles are supplied by the fifth cervical nerve, the levator usually receiving also a branch from the third cervical, and paralysis of them renders forced elevation of the scapula without rotation impossible. The diagnosis is difficult, except when there is coincident paralysis and atrophy of the trapezius, and in that case the patient is unable to draw the scapula towards the vertebral column; while paralysis of the levator anguli scapulæ is recognised by the inability to effect the characteristic elevation of the scapula.

(3) *Paralysis of the Latissimus Dorsi.*—This muscle is supplied by the long subscapular nerve, and paralysis of it is rare as an isolated affection, although common as a subordinate symptom of progressive muscular atrophy. The arm cannot be adducted with the usual amount of force, and the hand cannot be brought with the usual facility to the buttock.

(4) *Paralysis of the Inward and Outward Rotators of the Upper Arm.*—The outward rotators consist of the infraspinatus muscle, which is supplied by the suprascapular nerve, and the teres minor, supplied by the circumflex; when these are paralysed all the movements requiring outward rotation of the arm are rendered difficult or impossible. In writing and drawing, the formation of straight lines from left to right is in part dependent upon the contraction of the infraspinatus and teres muscles, so that these manual operations are rendered somewhat difficult. If the arm be rotated inwards, the patient is unable to rotate it outwards. When the muscles are atrophied, there is an increased depression of the infraspinatus fossa.

The inward rotators consist of the subscapularis, the teres major, and in part also the latissimus dorsi, which are all innervated by the subscapular nerves. When these are paralysed, all movements of the hand towards the opposite side of the body

or head are rendered difficult or impossible; and when the arm is rotated outwards the patient no longer retains the power to rotate it inwards, except perhaps to a limited degree by

FIG. 69.

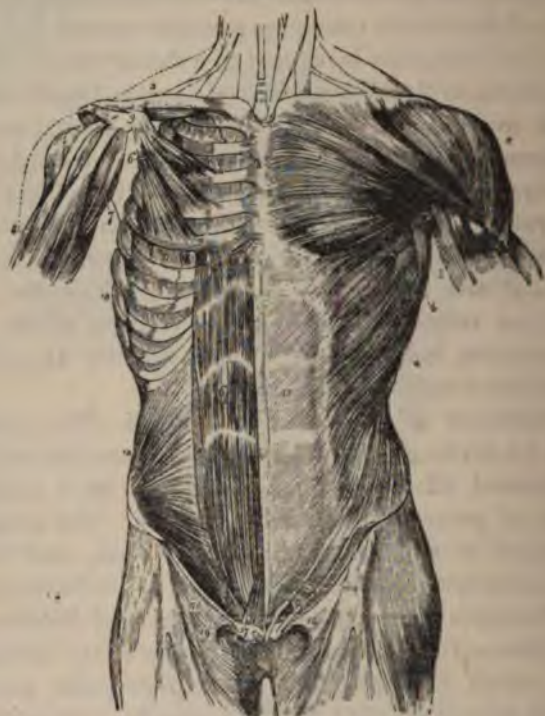


FIG. 69. *Muscles of the Anterior Aspect of the Trunk* (from Wilson).—On the left side of the body the superficial layer is seen, on the right the deeper layer.

- |   |  |
|---|--|
| 1, Pectoralis major.                    | left, the linea semilunaris; the       |
| 2, Deltoid.                             | transverse lines above and below       |
| 3, Anterior border of the latissimus    | the number, the lineæ transversæ.      |
| dorsi.                                  | 14, Poupart's ligament.                |
| 4, Serratus magnus.                     | 15, External abdominal ring.           |
| 5, Subclavius, right side.              | 16, Rectus muscle of the right side    |
| 6, Pectoralis minor.                    | brought into view by the removal       |
| 7, Coraco-brachialis.                   | of the anterior segment of its         |
| 8, Upper part of the biceps, showing    | sheath; * posterior segment of its     |
| its two heads.                          | sheath with the divided edge of        |
| 9, Coracoid process of the scapula.     | the anterior segment.                  |
| 10, Serratus magnus, right side.        | 17, Pyramidalis muscle.                |
| 11, External intercostal muscle of the  | 18, Internal oblique.                  |
| fifth intercostal space.                | 19, Conjoined tendon of the internal   |
| 12, External oblique.                   | oblique and transversalis.             |
| 13, Its aponeurosis: the median line to | 20, The lower curved border of the in- |
| the right of this number is the         | ternal oblique muscle.                 |
| linea alba; the curved line to its      |  |



means of the pectoralis major. The arm is maintained in a position of abnormal rotation outwards; but, contrary to what occurs when the outward rotators are contracted, passive mobility is preserved.

(5) *Paralysis of the serratus magnus muscle*, which is innervated by the posterior or long thoracic nerve, not unfrequently occurs as an isolated affection. In the great majority of cases the paralysis is peripheral, since the long course and comparatively superficial position of the nerve exposes it to *direct injury*, from carrying heavy loads on the shoulder, from pressure or contusion, blows, concussion of the shoulder, and gunshot wounds. Both unilateral and bilateral paralysis of the serratus have been observed after over-exertion of the muscles of the shoulder, as, for example, in mowers, puddlers, and other mechanics. In these cases it is probable that the nerve, as it perforates the scalenus medius, suffers injury when violent and repeated movements of the shoulder are performed. Paralysis of the serratus is also caused by exposure to cold; it is more frequent in men than in women, and may supervene as a sequel of typhoid fever. It may also occur as a symptom of spinal or cerebral disease, but in that case other muscles are coincidentally affected, especially the lower portion of the trapezius, the latissimus dorsi, the rhomboidei, &c.

*Symptoms.*—The paralytic symptoms are frequently preceded for some time by neuralgic pains in the neck and round the shoulder-blade (Poore), and some difficulty may be experienced in performing certain movements. When the muscle becomes paralysed, and the arm is hanging down by the side, the scapula is somewhat raised and approximated to the vertebral column, and so rotated on its axis that its inner or vertebral border is directed obliquely upwards and outwards, the inferior angle is drawn close to the vertebral column, and stands out slightly from the thoracic wall. These symptoms are caused by the unantagonised action of the rhomboidei, levator anguli scapulæ, trapezius, and pectoralis minor.

When certain movements are performed, very striking symptoms make their appearance on the side affected. The patient experiences some difficulty in raising the extended arm above the horizontal level. The reason of this is that the

rotation forwards of the scapula and the elevation of its external angle, which is mainly effected by the serratus, is much impaired, but as the rotatory action of the trapezius can still be brought into play, the arm can be raised to a level midway between the horizontal and vertical positions. Forcible rotation of the scapula forwards and fixation of the bone enables the arm to be raised to a vertical position. When the arm is raised to the horizontal position, the inner border of the scapula is drawn more and more inwards towards the vertebral column, pushing a mass of muscle before it; and if the paralysis be bilateral, the inner borders of the scapulæ may actually touch one another. If the raised arm be brought forward, the inner border of the scapula becomes more and more separated from the costal wall; so that a deep fossa is formed in which the hand may easily be laid and the inner surface of the bone felt. In bilateral paralysis the scapulæ enclose a deep hollow in which the muscular bellies of the rhomboidei distinctly project. If contraction of the muscle can be effected by faradic irritation of its nerves, the characteristic deformity is at once removed. Other movements are also interfered with. It is difficult to cross the arms in front of the chest, and to move the apex of the shoulder forwards as is required in delivering a blow in fencing, and the patient offers less resistance on the paralysed side to forcible retraction of the shoulder. The digitations of the serratus magnus with the external oblique are not observed on the paralysed side during forced inspiration.

Disturbances of sensibility are only rarely present; considerable *atrophy* of the affected muscle is usually observed in progressive muscular atrophy, as well as in severe traumatic neuritis, but it only occurs to a slight degree in paralysees of central origin, and in mild rheumatic cases.

In traumatic and severe rheumatic paralysees the reaction of degeneration is present; in paralysis from progressive muscular atrophy there is simple diminution of the electrical excitability; and in central paralysees and those produced by slight pressure, the excitability undergoes no change, or only a slight diminution. The course of paralysis of the serrati varies. Rheumatic paralysees and those arising from slight pressure almost always recover. Traumatic paralysees are usually of long duration, and



are not unfrequently incurable. The paralyzes arising from progressive muscular atrophy are also incurable. In protracted cases gradual contraction of the antagonist muscles takes place.

The following case of paralysis of the serratus magnus was kindly sent to me by Dr. Hardie, and I am indebted for the notes of the case to Mr. Challinor, who was at the time one of the house physicians at the Royal Infirmary. The accompanying engraving is from a sketch of the patient by Mr. Withers.

FIG. 70.



G. B., æt. 32 years, boiler-maker, entered the Royal Infirmary on December 24th, 1879. Five or six weeks before his admission, he was walking on a frosty morning on an inclined plank of wood, when his feet slipped, and he fell backwards and to the left. He threw out his right arm horizontally to protect himself, and the edge of the plank struck him below the right armpit and shoulder blade. He felt little or no pain at the time, and the skin over the part struck did not subsequently become discoloured. Some days after the accident he felt that he could not raise his right arm as well as usual, but he did not think there was anything seriously wrong until a fortnight ago, when a companion drew his attention to a deformity of his right shoulder blade.

*Present Condition.*—As the patient stands with his arms hanging by his side, the only noticeable deformity is that the inferior angle of the right scapula projects somewhat further from the costal wall, is somewhat nearer the middle line, and on a slightly higher level than the corresponding angle of the left scapula. The right scapula is also rotated, so that its internal border slants slightly upwards and outwards, its inferior border being more horizontal than that of the left. The superior internal angle of the right scapula is on a slightly higher level than that of the left, but the external angle with the shoulder of the right side appears to be on the same level as that of the left.

When the patient extends his arms horizontally outwards the right shoulder blade is drawn backwards and inwards, so that its internal border is parallel with and close to the spines of the dorsal vertebræ (*Fig. 70*). The internal border of the left scapula, on the other hand, slants obliquely from above downwards and from within outwards, the upper angle being close to the spines of the vertebræ, and the lower one  $4\frac{1}{2}$  in. removed from them. The right scapula is one inch higher than the left, and the internal border of the former stands out two inches from the costal wall, while the internal border of the left is closely applied to it.

On the arms being extended horizontally forwards the right scapula is drawn outwards and rotated on its vertical axis, so that it projects from the chest like a wing, the internal border being  $2\frac{1}{2}$  in. from the costal wall, whilst the corresponding border of the left is closely applied to it. The lower angle of the right scapula is  $1\frac{1}{4}$  in. from the middle line; that of the left  $6\frac{1}{2}$  inches. The upper and internal angle of the right is  $\frac{3}{4}$  in., and that of the left 2 in. from the spines of the vertebræ; while the right scapula stands also  $1\frac{1}{2}$  inches higher than the left.

On the arms being crossed in front, so that the tips of the fingers touch opposite shoulders, the deformity of the right shoulder-blade almost disappears, the inner border being closely applied to the costal wall. The lower angle of the right is  $4\frac{1}{2}$  in., and of the left  $5\frac{1}{2}$  in. from the middle line; and the internal superior angle of the right 3 in., and of the left  $3\frac{1}{4}$  in. from the middle line. The upper border of the right is one inch higher than that of the left.

When the patient raises the arms in a vertical direction the left becomes closely applied to the left ear, while the right is 7 in. removed from the right ear. The inferior angle of the right scapula is now  $2\frac{1}{2}$  in., and of the left  $7\frac{1}{2}$  in. from the spines of the vertebræ; while the inner border of the right scapula is  $2\frac{1}{2}$  in. from the costal wall, and the left closely applied to it; the inferior angles of both scapula being on the same level.

When the patient takes a deep inspiration, the digitations of the serratus magnus with the external oblique are plainly visible in the left side, but the wall of the chest remains quite smooth on the right side. During quiet respiration each half of the chest measures  $18\frac{1}{2}$  in.; on deep expiration the measurements are 18 in.; but on deep inspiration the left side measures  $19\frac{3}{4}$  in., and the left  $18\frac{1}{2}$  in. only.



When a faradic current is passed through the anterior fibres of the deltoid of the injured side, a deformity of the shoulder blade is produced similar to that caused when the arms are extended laterally at right angles to the body. When the current is passed through the anterior fibres of the left deltoid, the shoulder blade of that side assumes a position closely resembling that of the diseased side when the arms are hanging by the side. The rhomboidei muscles act energetically to a weak faradic current. No contraction of the right serratus magnus can be obtained by either current when applied cutaneously, but distinct contractions are obtained to a galvanic current from 20 cells Leclanché by means of electric acupuncture. He was ordered five grains of iodide of potassium to be taken three times a day, and the galvanic current to be passed through the paralysed muscle by means of electric acupuncture.

The patient remained a fortnight in the infirmary without presenting any notable signs of improvement, and then became an out-patient. Towards the end of January, the affected muscle began to contract decidedly during voluntary movements of the arm, but there was no manifest change in the electric reactions obtained. From this date rapid improvement took place; and I lost sight of him for a time, and did not see him until the month of May following, when he presented himself at

FIG. 71.

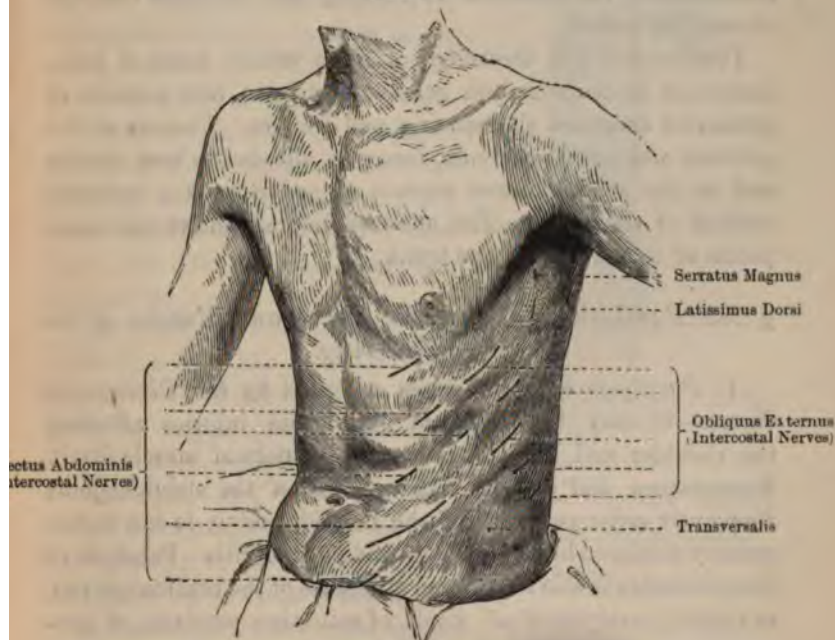


FIG. 71. Surface of Trunk

my rooms, and I could not detect a trace of deformity, but the right serratus magnus did not even then respond so readily to the faradic current as the corresponding muscle of the left side.

*Remarks.*—The paralysis in this case was not likely to have been caused by direct injury to the muscle, inasmuch as the blow received under the armpit during the fall appears to have been of a very trifling character. It is more probable that there was injury and consequent neuritis of the long thoracic nerve as it passes through the scalenus medius, caused by the sudden contraction of the muscle when the patient threw out his right arm in order to protect himself while falling. The deformities produced by passing a faradic current through the anterior fibres of the deltoid on the paralysed and sound sides show that contraction of these fibres contributes greatly in producing the deformities of the shoulder-blade on the affected side when the arm is horizontal. It is also probable that contraction of the pectoralis minor contributes in both instances in producing the deformity.

The diagnosis of paralysis of the serratus when uncomplicated presents no difficulty; it may be recognised by reference to the description of the symptoms. The prognosis is determined by the cause of the disease, the degree of the atrophy, the alterations of the electrical excitability and the time that the disease has lasted.

*Treatment.*—The treatment of these various forms of paralysis must be conducted on general principles, and consists of graduated exercises, shampooing, and friction. The use of the galvanic and faradic currents, however, affords the best results and in the severer forms electric acupuncture is a valuable method of treatment. The annexed diagram shows the motor points of the surface of the trunk.

§ 295. *Paralysis of the several Nerves and Muscles of the Upper Extremity.*

(1) *Paralysis of the Muscles supplied by the Circumflex Nerve.*—It may be paralysed by various injuries affecting the shoulder and shoulder-joint, or the deltoid muscle itself. Rheumatism and chronic inflammation of the shoulder-joint frequently cause paralysis of the deltoid, and it is not unfrequently produced by exposure to cold and neuritis. Paralysis of the circumflex is also a symptom of disease of the brachial plexus, of central paralysees of all kinds, of saturnine paralysis, of progressive muscular atrophy, and of pseudo-muscular hypertrophy.



The symptoms are almost exclusively those of paralysis of the deltoid muscle. The arm cannot be raised; and when attempts are made to raise it, the deltoid remains quite relaxed and the arm lies flat and immovable against the wall of the thorax. It is also impossible to raise the arm in a forward direction.

The muscle frequently atrophies and the shoulder-joint becomes so loose that a deep groove can be felt through the atrophied muscle, between the head of the humerus and the articular surface of the scapula. There may be pain in the shoulder-joint and in the substance of the muscle, but other sensory disturbances in the region of distribution of the circumflex nerve are rare.

The electric excitability may be normal at first, and then gradually undergo diminution, especially in progressive muscular atrophy, and in paralysis resulting from rheumatism of the shoulder-joint. The various phases of the reaction of degeneration may be present in the whole muscle, or limited to particular portions of it.

When the paralysis is persistent the atrophy gradually increases, the joint becomes loose, and in many cases ankylosis eventually takes place, the arm remaining more or less useless.

(2) *Paralysis in the Region of Distribution of the Musculo-Cutaneous Nerve.*—Paralysis of this nerve leads to impairment or complete impossibility of flexing the forearm on the upper arm, more especially when an attempt is made to bend the arm in a position of supination, inasmuch as in that position the flexor action of the supinator longus is no longer exerted. The seat of



FIG. 72. *Muscles of the Anterior Aspect of the Upper Arm (from Wilson.)*

- 1, Coracoid process of the scapula.
- 2, Coraco-clavicular ligament (trapezoid).
- 3, Coraco-acromial ligament.
- 4, Subscapularis.
- 5, Teres major.
- 6, Coraco-brachialis.
- 7, Biceps.
- 8, Upper end of the radius.
- 9, Brachialis anticus.
- 10, Internal <sup>triceps.</sup>

FIG. 73.

FIG. 73. *Superficial Muscles of the back of Forearm (from Wilson).*

- 1, Biceps.
- 2, Brachialis anticus.
- 3, Lower part of the triceps, inserted into the olecranon.
- 4, Supinator longus.
- 5, Extensor carpi radialis longior.
- 6, Extensor carpi radialis brevior.
- 7, Tendons of insertion of these two muscles.
- 8, Extensor communis digitorum.
- 9, Extensor minimi digiti.
- 10, Extensor carpi ulnaris.
- 11, Anconeus.
- 12, Flexor carpi ulnaris.
- 13, Extensor ossis metacarpi and extensor primi internodii pollicis lying together.
- 14, Extensor secundi internodii pollicis.
- 15, Posterior annular ligament. The tendons of the common extensor are seen on the back of the hand, and their mode of insertion on the dorsum of the fingers.

FIG. 74.

FIG. 74. *Deep Muscles of the back of Forearm (from Wilson).*

- 1, Humerus.
- 2, Olecranon.
- 3, Ulna.
- 4, Anconeus.
- 5, Supinator brevis.
- 6, Extensor ossis metacarpi pollicis.
- 7, Extensor primi internodii pollicis.
- 8, Extensor secundi internodii pollicis.
- 9, Extensor indicis.
- 10, First dorsal interosseous muscle. The other three dorsal interossei are seen between the metacarpal bones of their respective fingers.



the lesion may often be ascertained by the presence of anæsthesia along the radial border of the forearm. This form of paralysis is usually associated with paralysis of other muscles innervated by branches from the brachial plexus, and is rare as an isolated affection.

(3) *Paralysis in the Region of Distribution of the Musculo-Spiral Nerve.*—The musculo-spiral nerve is more frequently affected than any other branch of the brachial plexus, probably owing to its exposed position as it winds round the upper arm. Paralysis of this nerve is supposed to be frequently caused by exposure to a draught of cold air, or sleeping on damp earth; but it is more probable that in the majority of these cases there is compression of the nerve. This occurs very commonly during deep and prolonged sleep, especially in states of intoxication; and under these circumstances the paralysis appears when the patient awakes. The nerve may also be subjected to compression by improperly constructed crutches, and in various other ways which need not be detailed. Rheumatic paralysis of the musculo-spiral is by no means so frequent as was at one time believed. Paralysis of the nerve is caused by various wounds, contusions, and other injuries, and may result from neuritis. Hysterical paralyses of the musculo-spiral are rare. This nerve is frequently implicated in central and especially in cerebral paralysis. Lead poisoning is a frequent cause of paralysis of the musculo-spiral nerve, the affection being usually preceded by colic and arthralgia. Lead paralysis usually commences in the muscles supplied by the musculo-spiral in the forearm, and especially the *extensor communis digitorum*, but ultimately the muscles of the hand, upper arm, and shoulder, as well as those of the lower extremities, are not unfrequently affected.

*Symptoms.*—When the musculo-spiral nerve is completely paralysed, the hand is kept in a state of flexion, it hangs flaccid and cannot be raised or extended; the thumb is flexed and adducted, and the fingers are flexed over the thumb. When an attempt is made to extend the fingers, the *interossei* and *lumbricales* alone act; and these only extend the two terminal phalanges, whilst they flex the basal phalanx. The thumb and index finger cannot be abducted or extended. There is inability to supinate the forearm, especially when it is extended so as to

exclude the action of the biceps; nor can it be bent and half supinated by the supinator longus. Paralysis of the supinator longus is readily recognised by requesting the patient to make a powerful effort to flex the arm against a resisting object when the arm is maintained in a half-flexed position, and midway between pronation and supination, the muscle does not become rigid, but remains flaccid and soft. If the triceps be simultaneously affected, the patient cannot extend the arm with any degree of force. If the hand be laid upon a table, no lateral movements can be made with it, nor can it be raised from the surface of the table; but the lateral movements of the fingers remain unimpaired. The action of the flexors is apparently weakened; but the feebleness of their contractions is due to the fact that the flexed position assumed by the hand approximates the points of origin and insertion of the flexors.

More or less anæsthesia usually accompanies motor paralysis of the musculo-spiral nerve, and if the cause be situated high up the anæsthesia affects the region of the superior and inferior external cutaneous branches; but if lower down, the dorsal surfaces of the first three and a half fingers, as far as to the second phalanx, and the corresponding parts of the back of the hand and ball of the thumb, are alone affected.

The extensor muscles are frequently atrophied, and sometimes a painless swelling of the extensor tendons over the wrist-joint is observed. This swelling has been described under the name of *tenosynitis hyperplastica*, and is caused by the irritation to which the tendons are exposed while running over the strongly-flexed wrist. At times it may be the result of trophic disturbances from the paralysis of the nerve (Erb).

The electric excitability is normal in the slight cases arising from pressure and exposure to cold. The seat of the lesion may sometimes be accurately ascertained by means of the electrical reaction, since while the excitability of the nerve may be normal below the lesion, no reaction can be obtained when the current is applied above it. The reaction of degeneration occurs in all severe traumatic paralyses and in lead paralysis, and the muscles present a high degree of atrophy.



*Diagnosis.*—Paralysis from compression is characterised by paralysis of *all* the muscles on the extensor side of the forearm, non-implication of the triceps, disturbance of sensibility *only* in the hand, and the persistence of normal electrical excitability. *Crutch* paralysis is characterised by implication of the triceps, by normal electrical reaction, and by exposure to this cause.

In *severe traumatic* paralysis different muscles will be affected according to the position of the wound, and the reaction of degeneration and atrophy of the affected muscles are always present.

*Lead paralysis* usually begins in the extensor communis digitorum, a few fasciculi of which are first affected, and then the whole muscle. The radial and ulnar extensors, and the extensors of the thumb, are then successively affected. The supinator brevis is only affected at a late period of the disease; and the supinator longus almost invariably escapes, being only affected in very rare cases, and then at a late stage and to a very moderate degree, and when the paralysis extends to the muscles of the upper arm. Both arms are usually affected shortly after one another. The reaction of degeneration appears early and very decidedly in a part of the paralysed muscles, and as usual it is associated with progressive atrophy of the muscle. In many cases the veins of the forearm, especially those of the extensor side, are remarkably swollen and varicose; the presence of the blue line on the gums will aid the diagnosis.

The *course* and *prognosis* of the disease depend upon the nature of the cause. Rheumatic paralysis often lasts from four to six weeks, and not unfrequently for months or even years; but recovery is almost sure to follow eventually. Recovery usually takes place in *crutch paralysis*, under appropriate treatment in from one to two weeks. Severe traumatic paralyses are as tedious and protracted here as elsewhere, and frequently from half a year to a year elapses before recovery is complete; but, unless an irreparable injury has been done to the nerve, recovery ultimately occurs.

Lead paralysis is always slow, months usually elapsing before the return of voluntary motion in the paralysed muscles.

Hysterical paralysis of the musculo-spiral varies very much in its course, like hysterical paralysis in general. Extension of the wrist, fingers, and thumb is the last movement which such patients recover.

FIG. 75.

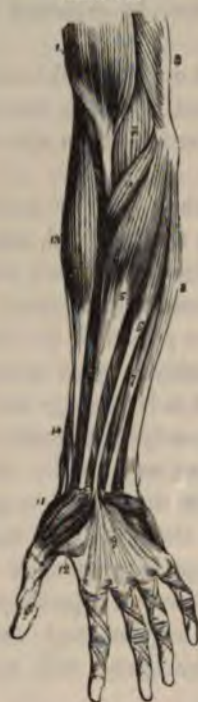


FIG. 75. *Superficial Muscles of the Fore-arm* (from Wilson).

- 1, Biceps, with its tendon.
- 2, Brachialis anticus.
- 3, Part of triceps.
- 4, Pronator radii teres.
- 5, Flexor carpi radialis.
- 6, Palmaris longus.
- 7, Part of the flexor sublimis digitorum; the rest of the muscle is seen beneath the tendons of the palmaris longus and flexor carpi radialis.
- 8, Flexor carpi ulnaris.
- 9, Palmar fascia.
- 10, Palmaris brevis.
- 11, Abductor pollicis.
- 12, Flexor brevis pollicis.
- 13, Supinator longus.
- 14, Extensor ossis metacarpi and extensor primi internodii pollicis, curving round the lower border of the forearm.

(4) *Paralysis in the Region of Distribution of the Median Nerve.*—Independent affection of the median nerve is rare, especially that due to rheumatism. Traumatic paralysis, on the other hand, is more frequent, either affecting the nerve above the arm or its terminal branch above the wrist. Some of the muscles supplied by the median are implicated in progressive muscular atrophy, especially those of the thenar eminence.

The symptoms of paralysis of the median nerve are impossibility of flexing the second phalanx in every finger, the third phalanx of the index and middle fingers, as well as flexion and opposition of the thumb. The power of abduction of the thumb is impaired. Flexion of the first with extension of the second and third phalanges, in the case of all the four fingers, can be easily effected by means of the interossei; and, indeed, there may be hyperextension of the two last phalanges, owing to the unantagonised action of these muscles. The patient is unable to perform any of the more



delicate movements of the thumb, which is permanently extended, adducted, and kept closely applied to the forefinger, as in the hand of the ape.

Owing to the deficient action of the flexor carpi radialis, flexion of the wrist is also accompanied by adduction, this being principally due to the action of the flexor carpi ulnaris. Pronation of the hand is almost impossible, but it can be performed in an incomplete manner by the supinator longus when the forearm is strongly supinated. The two ulnar fingers can still be partially bent, since the flexor profundus digitorum is in part supplied by the ulnar nerve. The muscles of the forearm and ball of the thumb frequently atrophy.

Disturbance of sensibility may be entirely absent, even when the median is completely divided above the wrist, no doubt in consequence of the anastomoses which the median forms with the other nerves of the forearm. If disturbances of sensibility be present, they are exhibited in the lateral part of the palm of the hand, on the palmar side of the thumb, index and middle fingers, and in the ungual phalanges on the dorsal side also.

Trophic disturbances of the skin and nails, such as glossy fingers, ulceration, pemphigus

FIG. 76.

FIG. 76. *Deep Muscles of the Forearm* (from Wilson).

- 1, Internal lateral ligament of the elbow joint.
- 2, Anterior ligament.
- 3, Orbicular ligament of the head of the radius.
- 4, Flexor profundus digitorum (the lumbricales removed).
- 5, Flexor longus pollicis.
- 6, Pronator quadratus.
- 7, Abductor pollicis.
- 8, Dorsal interosseous of the middle, and palmar interosseous of the ring finger.
- 9, Dorsal interosseous muscle of the ring finger, and palmar interosseous of the little finger.

vesicles, and abnormal growth of hair, not unfrequently make their appearance in paralysis of the median. The electrical reactions are the same as in paralysis of other nerves.

(5) *Paralysis in the Region of Distribution of the Ulnar Nerve.*—Although the nerve, from its superficial position in the upper arm and above the wrist, is much exposed to injury, it is not very often affected with paralysis. The most frequent causes of paralysis of this nerve are pressure, contusion, gunshot and other wounds, fractures of the humerus, dislocations of the shoulder, pressure of crutches, and sleeping upon the arm placed beneath the head. Duchenne saw paralysis of this nerve frequently in workmen who rest the elbow firmly on a hard support in carrying on their ordinary occupation. Progressive muscular atrophy affects by preference the small muscles of the hand which are supplied by the ulnar. (Table II., 1, 2, 3.)

The symptoms of paralysis of the ulnar are limitation of the power of ulnar flexion and adduction of the hand; difficulty or

FIG. 77.

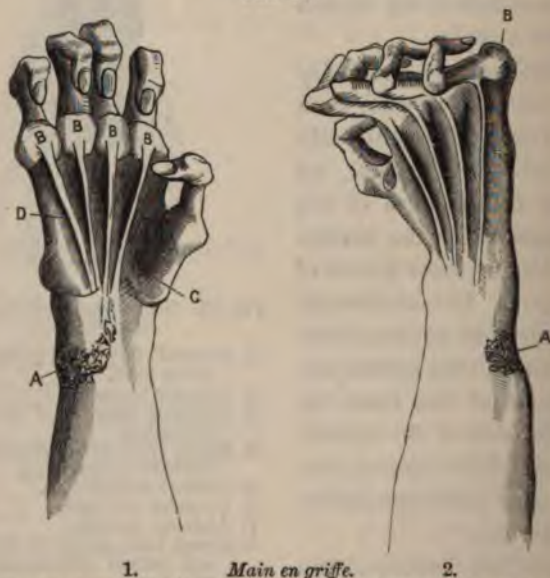


FIG. 77 (after Duchenne). (1) *Hand, Palmar surface.* (2) *Dorsal surface.*—A, Wound of the ulnar nerve; B, Ends of the metacarpal bones; D, Tendons of the flexor sublimis; C, Muscles of the ball of the thumb.



impossibility of completely flexing the two last fingers; loss of power of moving the little finger, of separating and compressing the fingers laterally against one another, and of the power of flexing the first and extending the second and third phalanges of all the fingers owing to paralysis of the interossei. If the interossei and lumbricales are alone paralysed, the traction of the extensor communis and of the flexors produces extension of the first and flexion of the two last phalanges, giving to the hand the claw-like appearance, which is so characteristic of paralysis of the ulnar nerve above the wrist, and of certain cases of progressive muscular atrophy. The patient is

FIG. 78.

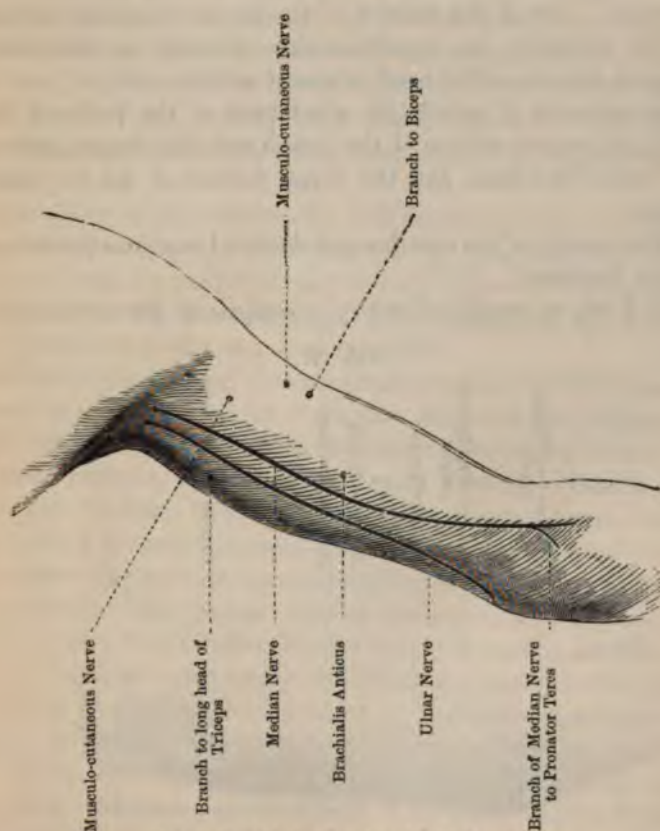


FIG. 78. Anterior Surface of Left Arm.

also unable to adduct the thumb and apply it firmly to the metacarpus of the index finger. In severe and protracted cases the first phalanges are dislocated backwards upon the metacarpal bones by the unantagonised action of the extensor communis digitorum; while the second become dislocated forwards on the first phalanges, and the third on the second by the unantagonised actions of the flexor digitorum sublimis and profundus respectively. The most characteristic form of the claw hand (*main en griffe*) is then produced.

The use of the hand in paralysis of the ulnar is not entirely abolished, inasmuch as motor power is preserved in the muscles supplied by the median nerve, although all the delicate movements required for writing or drawing or playing the piano are impaired. But if the muscles of the thenar eminence or part of the extensors be simultaneously affected, as frequently happens, the use of the hand is almost entirely lost.

Disturbances of sensibility affect part of the palm of the hand, the palmar surface of the fourth and fifth fingers, part of the back of the hand, and the dorsal surface of the two ulnar fingers.

The atrophy of the muscles and electrical reactions present no special features.

(6) *Various combinations of paralysis of the nerves of the*

FIG. 79.



FIG. 79. Posterior Surface of Left Arm.



*upper extremity* occur, and each case must be specially investigated as it presents itself. The paralyses which occur after dislocation of the shoulder-joint manifest a great variety. In subarachnoid luxations the subjacent nerve trunks of the brachial plexus are especially liable to injury, and the whole of the nerves may be compressed or lacerated, or may be only slightly, or not at all, damaged, according to circumstances. Sometimes the circumflex, musculo-cutaneous, and the three nerve trunks of the forearm are equally affected; at other times, only the circumflex and musculo-spiral nerves are implicated; and many other variations may occur. Recovery is generally slow, and the reaction of degeneration is exhibited in the affected nerves and muscles.

In *paralysis from fracture of the humerus*, the result depends upon whether one or several nerve trunks of the forearm have been injured, or are subsequently implicated in the development of the callus. The musculo-spiral is most frequently paralysed, then the ulnar, and more rarely the median nerve.

Dislocations of the elbow-joint and fractures in its vicinity, and of the forearm, are often followed by paralysis; and the ulnar and median nerves are particularly liable to be affected. Paralysis may also be caused by tight bandaging; all these paralyses are obstinate, and often incurable.

Rheumatic paralyses of the arm present curious combinations. Erb mentions cases in which there was simultaneous paralysis of the deltoid, biceps, brachialis anticus, and supinator longus; and, as he remarks, the seat of the injury must have been at a point where the fibres forming the circumflex, musculo-cutaneous, and a part of the musculo-spiral nerves lie in close proximity to each other. The muscles were notably atrophied, and presented the reaction of degeneration. Similar cases have been described by Remak and Hoedemaker. Nearly the same muscles are paralysed in the form of paralysis which Duchenne has described in newly-born children under the name of "*paralysie obstétricale infantile du membre supérieur*." This paralysis always occurs in children whose birth had been effected by turning, or some other operative procedure, and it no doubt results from mechanical compression applied during delivery. In these cases

the arm hangs immovable by the side of the body, is rotated inwards, persistently extended, and the child is unable to flex the forearm or to raise the arm; but the movements of the hands and fingers are preserved. The deltoid, brachialis anticus, infraspinatus, the teres minor, and probably the supinators, are paralysed. The reaction of degeneration is usually present, and the prognosis is unfavourable. A variety of paralysis has been described by Straus, which implicates all the muscles of the superior extremity except those innervated by the median nerve. The patient on waking in the morning complains of



FIG. 80. Anterior Surface of Left Forearm.



formation and weakness of one of the upper extremities. The weakness extends rapidly until all the muscles innervated by the brachial plexus, except those supplied by the median nerve, are completely paralysed. The sensory disturbances also increase until there is complete anæsthesia of the cutaneous surface of the upper extremity, except the portions innervated by the median nerve. The electric contractility of both muscles and nerves remains unaffected, and complete recovery takes place at the end of about seven weeks under proper treatment. Other forms of paralysis of the forearm may be caused by injuries, such as dislocations, fractures, or compression of the forceps.

FIG. 81.

FIG. 81. *Posterior Surface of the Left Forearm.*

*Treatment of the Paralyses of the Upper Extremity.*—The removal of the cause must be attempted, and surgical means are not unfrequently successful. In rheumatic paralysis counter-irritants, diaphoretics, and the iodide of potassium may be tried. When neuritis is present antiphlogistics and the application of galvanic currents are useful.

In hysterical and central paralysis the primary disease must be treated. The treatment of lead paralysis will be mentioned in a subsequent section. In chronic cases of traumatic paralysis and in paralysis resulting from articular rheumatism, or from chronic neuritis, improvement may be obtained by the employment of malt and mud baths, and the baths of Wildbad, Teplitz, Wiesbaden, &c. Electricity is useful in the treatment of all the forms of paralysis. In severe traumatic paralysis long-continued and repeated applications of the galvanic current are requisite. In slight paralysis from compression faradisation is tolerably successful. The cervical portion of the spinal cord may be galvanised, and it is probable that galvanisation of the sympathetic also may be useful. Active and passive gymnastic exercises frequently aid and hasten the recovery.



## CHAPTER VII.

## DISEASES OF THE DORSAL NERVES AND LUMBAR PLEXUS.

## (I.)—DISEASES OF THE DORSAL NERVES.

§ 296. *Dorso-Intercostal Neuralgia.*

THIS form of neuralgia is situated in the region of distribution of the sensory branches of the twelve pairs of dorsal nerves. The anterior branches of the nerves usually suffer, giving rise to true intercostal neuralgia, in which the skin of the whole of the anterior and lateral wall of the thorax and abdomen is affected down to the symphysis pubis. When the posterior branches are implicated, the pain affects the skin of the back and loins as far down as the crista ili. The second and third intercostal nerves supply cutaneous branches to the axilla and to the inner surface of the upper arm, and these parts may occasionally be the seat of neuralgia. Dorso-intercostal neuralgia is generally unilateral, and the left side is most commonly affected. The fifth to the ninth nerves are those most frequently affected, and generally only one or two nerves are implicated. This form of neuralgia may be combined with brachial and lumbo-abdominal neuralgia, or with angina pectoris.

*Etiology.*—Women are specially liable to dorso-intercostal neuralgia. It comes on usually between the ages of twenty and forty, in consequence of over-suckling, or the exhaustion caused by menorrhagia or leucorrhœa, and attacks weak, nervous, hysterical, and anæmic subjects.

The exciting causes are exposure to cold, and injuries of various kinds, while in other cases the neuralgia is a symptom of disease of the nerves, such as neuritis                      mata. Disease

in the neighbourhood of the nerves may also give rise to intercostal neuralgia. The most usual diseases with which it is associated are aortic aneurisms, disease of the vertebræ or of the ribs, pulmonary phthisis, and dilatation of the venous plexus in the interior of the vertebral canal. Diseases of the spinal cord, especially circumscribed myelitis, spinal meningitis, tumours in the vertebral canal and spinal cord, and locomotor ataxy, are frequently associated with intercostal neuralgia.

*Symptoms.*—The pain of intercostal neuralgia is dull, tensive, and continuous, but it is usually interrupted by tearing, lancinating, burning pains, which may increase to a regular paroxysm. All violent respiratory movements, such as sneezing, aggravate the pain, so also does slight pressure on the skin, as that of the bed clothes, but steady firm pressure often relieves it.

*The painful points* are : (1) *A vertebral point* close to the vertebral column, where the nerve emerges from the intercostal foramen ; (2) *a lateral point*, where the lateral perforating branch becomes subcutaneous, midway in the intercostal space ; and (3) *an anterior or sternal point*, where the anterior perforating branch pierces the muscles close to the sternum and in the abdomen over the rectus muscle. The whole length of the intercostal nerves and several of the spines of the corresponding vertebræ are frequently extremely sensitive and tender.

The pain not unfrequently radiates towards the back and arm or into the loins or lower extremities. *Hyperæsthesia* of the affected skin is common ; and *anæsthesia*, although less common, has been observed, usually limited to a small circumscribed area. Motor phenomena are generally observed which interfere to some extent with respiration.

With respect to vaso-motor and trophic disturbances, herpes zoster is the best known, although its relations to neuralgia are by no means constant. Herpes zoster is seldom attended by severe neuralgia in young persons, but in old persons the neuralgia generally precedes and outlasts the herpes.

The course of intercostal neuralgia is irregular, but it is usually obstinate, and recovery is very gradual. In central disease of the nervous system, recovery is slow, and the patient is liable to relapses ; while in such diseases as pulmonary phthisis and disease of the vertebræ, the neuralgia generally terminates only with death.



*Diagnosis.*—The diagnosis of *dorso-intercostal* neuralgia is surrounded with considerable difficulties. It is liable to be confounded with myalgia, either pleurodynia or lumbago. The seat of the pain in certain muscles, its aggravation on making certain movements and on pressure, the absence of painful points, and the rapid recovery in the course of a few days, serve to distinguish pleurodynia and lumbago from intercostal neuralgia. Diseases of the thoracic organs must be distinguished from neuralgia by physical examination. Angina pectoris resembles intercostal neuralgia in some respects; but the intense anxiety and feeling of impending death, along with the characteristic radiation of the pain, should prevent the diseases from being confounded.

The prognosis will depend upon the cause of the disease.

*Treatment.*—The general principles of treatment are the same as for other forms of neuralgia. Counter-irritation, especially in the form of flying blisters, is very valuable in the treatment of this variety. They should be applied in succession over the painful points. In slight cases the milder cutaneous irritants and anodyne embrocations are useful. Subcutaneous injection of morphia is, as usual, in neuralgia, a valuable agent in the treatment.

*Mastodynia.*—Neuralgia of the female breast forms a special variety of intercostal neuralgia. The skin over the mammæ is supplied by the lateral and anterior perforating branches of the second to the sixth intercostals, and by minute branches of the supra-clavicular nerves; whilst the proper substance of the gland is supplied by the lateral perforating branches of the fourth, fifth, and sixth intercostals. Neuralgia of the breast may appear in those who have a strong neurotic tendency, along with the first development of the breasts at puberty, especially in cases when puberty is prematurely developed in consequence of self-abuse. Neuralgic pain may come on during pregnancy, although a large proportion of the pains felt in the mammary gland during this period are caused by mechanical distension of the breast. Neuralgia frequently follows shrinking of the nipples; and the irritation of cracked nipples may be the exciting cause of attacks of the disease. Anæmia, chlorosis, and hysteria also take a great part in the production of the disease.

Injuries of the gland, neuromata, or painful tubercles of the nerves, may also be the starting points of neuralgia. The pain of "irritable" breast is very violent, and described as tearing, cutting, boring, and lancinating; and appears in paroxysms, which are usually of short duration, but may last several hours. The breast feels heavy, and the patient cannot lie on the affected side; the slightest contact, even the pressure of the clothes, is unbearable.

*Painful points* may be found on the nipple or on the sides of the breast, but they are indefinite; and the spinous processes of the second to the sixth dorsal vertebræ are usually tender on pressure. There is generally a great deal of hyperæsthesia, and the paroxysms are sometimes accompanied by vomiting. The pain radiates into adjoining regions, and the severity of the paroxysms is increased during the catamenial period.

*Treatment.*—The general principles of treatment of dorso-intercostal neuralgia are the same as for other forms of the disease. Counter-irritation, especially in the form of flying blisters, is very valuable. In slighter cases the milder cutaneous irritants and anodyne embrocations are useful. Subcutaneous injection is, as usual, a valuable agent in the treatment. Electricity is very useful. The faradic current may be used as a cutaneous irritant in the form of the brush or moxa. In using the galvanic current, the cathode should be placed on the vertebral column, and the anode upon the lateral and anterior painful points; the current should be strong and stable.

When herpes zoster is associated with neuralgia, the surface should be covered by some indifferent and protective ointment or plaster, to which some narcotic may be added. Cotton wool also makes an admirable protective covering. Flying blisters may be applied to the side of the vertebral column, as recommended by Dr. Anstie; but in a case in which a woman took the treatment out of my hands and applied a large blister over the vesicles, complete and immediate relief was afforded.

The treatment of mastodynia is not very successful. The causes of the disease, as anæmia and disturbance of the generative organs, must be combated. A belladonna plaster may be found useful along with the usual remedies, as anodynes,



electricity, and tonics. The breast may be enveloped in cotton wool, fur, or any other soft and warm covering. Removal of painful indurations and amputation of the breast should only be undertaken as a last resort.

§ 297. *Paralysis of the Dorsal Muscles.*

Paretic and paralytic conditions of the extensors of the back are not uncommon. In youth various degrees of weakness of the dorsal muscles are often present, sometimes on one and sometimes on both sides, giving rise to various forms of spinal curvature. Rheumatic affections may cause paralysis of one or several of the dorsal muscles, and the same result may supervene upon injuries of the back. Paralysis of the dorsal muscles is rare in cerebral disease, but is not unfrequent in spinal affections. Progressive muscular atrophy not unfrequently extends to the dorsal muscles, and weakness of the long extensors of the back forms a characteristic feature of pseudo-hypertrophic paralysis. Occasionally weakness with atrophy of these muscles occurs in young persons without any obvious cause.

The muscles principally affected are the sacro-lumbalis and the longissimus dorsi, with their continuations towards the neck and head, and the small muscles between the several vertebræ. We ought to determine clinically whether the paralysis has its seat in the lumbar, dorsal, or cervical portion. When the extensors of the dorsal region on both sides are paralysed the vertebral column forms a large and equable curve, the patients appear bent and doubled up as in old age, and are unable to hold themselves erect; but passive straightening of the vertebral column can be effected with tolerable facility, and this distinguishes paralytic kyphosis, as the condition is called, from kyphosis, the result of muscular contracture or disease of the vertebræ. If the paresis or paralysis be unilateral, various forms of paralytic scoliosis are produced.

Paralysis of the extensors in the *lumbar region* presents very characteristic features. The lumbar vertebræ are curved inwards so as to form a remarkable hollow in the back, a hollow which is increased by the upper part of the body being thrown backwards in order to compensate for the incurvation of the

lumbar portion. A plumb line allowed to drop from the most prominent spinous process of the dorsal vertebræ clears the sacrum generally by one to one and a half inches. The further peculiarities of this variety of paralysis will be described when we come to discuss pseudo-hypertrophic paralysis.

If the posterior *muscles of the neck* are alone paralysed, the head can no longer be carried erect, but the patient can raise it by a peculiar swinging movement, and he then usually carries it inclined backwards, supported only by the anterior muscles of the neck.

*Paralysis of the abdominal muscles* is very rare as an isolated affection, but is a common symptom of spinal paralysis, and occurs occasionally in progressive muscular atrophy. When the paralysis is unilateral, the umbilicus is carried to the sound side with each movement of forcible expiration. When the paralysis is bilateral, there is great weakness of expiration, and of all expiratory reflex acts, such as coughing, sneezing, &c.

The power of compressing the abdomen is impaired, causing difficulty of evacuating the contents of the rectum and bladder. The abdomen is large and protuberant, and its walls relaxed, the patient is unable to raise the upper part of the body from the recumbent position, or to sit in bed without being propped up by the hands. In walking or standing the upper part of the body is bent slightly forwards and balanced exclusively by the lumbar muscles; hence each shifting of the centre of gravity backwards renders the patient liable to fall backwards, because the abdominal muscles are incapable of drawing the trunk forwards. When paraplegia is present this symptom cannot be determined.

*Treatment.*—The treatment must depend on the nature of the primary disease, of which the paralysis is but a symptom. Electrical treatment is of great advantage, and both the galvanic and faradic currents may be useful.

When the paralysis is incomplete, benefit is sometimes obtained by a system of gymnastics; and in incurable cases appropriate orthopædic apparatus may be employed to replace the deficient muscular action. The employment of embrocations, baths, douches, liniments, change of air, and general tonic treatment is productive of good results.



## (II.)—DISEASE IN THE REGION OF DISTRIBUTION OF THE LUMBAR NERVES.

§ 298. *Lumbar Neuralgia.*

This form of neuralgia includes all varieties having their seat in the region of distribution of the first four pairs of lumbar nerves. The following are the regions supplied by these nerves:—The region of the loins, supplied by the posterior branches of these nerves; the gluteal region, inguinal region, hypogastrium, and mons veneris, part of the scrotum in the male, and of the labia majora in the female, the anterior lateral and median surface of the thigh, the anterior region of the knee-joint, the median surface of the leg, and the inner border of the foot as far as to the great toe. It is very rare for the whole of the branches of the lumbar plexus to be implicated; and, as a rule, only one or a few branches are affected. The different forms of the disease may be divided into two groups; the first of which includes neuralgia of the “short” nerves of the lumbar plexus, and which from its situation may be called *lumbo-abdominal neuralgia*; the second including neuralgia of the “long” nerves of the plexus, may be termed *femoral neuralgia*.

*Etiology.*—The causes of this form of neuralgia are not well known. Dr. Anstie mentions a well-marked instance of the disease which was excited by sudden fright in a woman exhausted by leucorrhœa, and with a decided neuropathic history. Exposure to cold, injuries, neuromata, and other tumours, compression of nerves from herniæ, or from accumulation of fæces, cancer in the pelvis or the vertebral column, diseases of the vertebræ, psoas abscesses, diseases of the uterus and vagina, have each been ascertained to have formed the exciting cause of the disease. Spinal diseases, as meningitis, myelitis, and tabes dorsalis, are also causes of the disease; and the pain of the knee in coxitis is a neuralgia caused by reflex irritation.

*Symptoms.*—(1) *Lumbo-abdominal Neuralgia.*—There is pain in the loins extending over the crista ilii as far as the buttock, with pain in the hypogastrium, mons veneris, and





The Painful Points are: (1) *Vertebral points*, corresponding to the posterior branches of the respective nerves; (2) *Iliac point*, corresponding to the middle of the crista ili; (3) *Abdominal points* above the symphysis pubis at the side of the linea alba; (4) *An inguinal point* in the groin, near the exit of the spermatic cord, from whence the pain radiates along the latter; (5) *A scrotal or labial point* situated in the scrotum or labium majus. The pain frequently radiates into neighbouring nerve territories, and especially into those supplied by the crural and intercostal nerves; and it is probable that the sympathetic plexuses of the pelvic organs are often implicated. Spasm of the cremaster, vomiting, herpes, increased sexual desire, with priapism and ejaculation of seminal fluid, may be mentioned as the most usual of the concomitant symptoms of this variety of neuralgia.

(2) *Femoral Neuralgia* may be subdivided into three varieties: (a) Neuralgia of the lateral cutaneous nerve of the thigh; (b) crural; and (c) obturator neuralgia.

(a) *Neuralgia of the Lateral Cutaneous Nerve of the Thigh*. The pain extends down along the outer and part of the posterior aspect of the thigh as far as the knee. A constant

GC, Genito-crural nerve.

G, Genital branch to spermatic cord or round ligament.

2, Muscular branch to cremaster.

C, Crural branch, cutaneous to surface of upper part of front of thigh.

EC, External cutaneous.

P, Posterior branch cutaneous to upper and outer part of thigh.

A, Anterior branch cutaneous to front of thigh.

ps, Muscular branches to psoas muscle.

AC, Anterior crural nerve.

3, Muscular branches to iliatus.

3', " " sartorius.

3", " " pectineus.

fa, Branch to femoral artery.

MC, Middle cutaneous to front of thigh.

IC, Internal cutaneous to inner part of thigh and leg.

LS, Internal or long saphenous.

a, Cutaneous over inner ankle.

f, " " to inner side of foot.

4, Muscular branch to rectus femoris.

4', " " vastus externus.

4", " " crureus.

4''' " " subcrureus.

4"" " " vastus internus.

Kj, Branch to knee-joint.

O, Obturator nerve.

hj, Branch to hip-joint.

c, Communicating with branches of internal cutaneous and internal saphenous.

5, Muscular branch to pectineus.

5', " " obturator externus.

6, " " adductor longus.

6', " " gracilis.

6'' " " adductor brevis.

6''' 6"" " " adductor magnus.

Kj', Branch to knee-joint.

L, Communicating branch to fifth lumbar nerve.

painful spot is present over the anterior superior spinous process of the ilium, where the nerve emerges from the pelvis, and less constant spots may be present along the outer side of the thigh.

(b) *Crural Neuralgia*.—The middle and inner part of the anterior surface of the thigh, the anterior surface of the knee, the inner surface of the leg, and of the foot as far as to the great toe are affected.

The painful points associated with this variety are : (1) One in the fold of the groin when the nerve emerges from the pelvis ; (2) the inner side of the knee-cap when the saphenous nerve appears beneath the skin ; (3) in front of the ankle-joint ; (4) at the base of the great toe.

Amongst the concomitant symptoms may be mentioned hyperæsthesia of the skin, especially in the vicinity of the knee-joint, anæsthesia of part of the surface is not unfrequent, and a feeling of numbness or formication in the region of distribution of the saphenous nerve. There may be also weakness and paresis of the muscles of the thigh, and the patient complains of weariness.

(c) *Obturator Neuralgia*.—The pain in this variety is confined to the inner side of the thigh, extending as far as the knee-joint. Formication on the inner surface of the thigh, and a feeling of stiffness and immobility of the adductors, are usually present. This form of neuralgia is associated with obturator hernia ; and Romberg has pointed out that when obturator neuralgia is present along with symptoms of strangulated intestines, obturator hernia may safely be inferred.

The course of all the varieties of femoral neuralgia is, as a rule, favourable, and its duration short, except in cases associated with grave organic disease.

The *diagnosis* is not unattended with difficulty. It may be confounded with myalgia, especially with lumbago. In the latter disease the pain is usually circumscribed, and does not radiate in various directions ; it is generally aggravated by stretching the body or raising a weight, and disappears when rest in the recumbent posture is maintained. The pain of renal calculi often cannot be distinguished from neuralgia, and the diagnosis from hip and knee-joint diseases requires care and circumspection.

*Treatment*.—The treatment of this form of neuralgia presents nothing special. Flying blisters, injection of morphia or



of atropine, and electricity, appear to give the best results. A descending stable current may be made to pass from the lumbar region of the vertebral column through the affected nerve.

§ 299. *Neuralgia Pudendo-Hæmorrhoidalis, and Neuralgia of the External Generative Organs generally.*

The external generative organs and neighbouring parts are supplied by the following sensory nerves. The external organs of generation receive their chief nervous supply from the sacral plexus by means of the dorsal and superficial perinæal branches of the internal pudic nerve, and the inferior or long pudendal branch of the small sciatic nerve; the integument in the region of the anus is supplied by the inferior hæmorrhoidal branches of the pudic nerve, and by branches from the fourth and fifth sacral nerves. The lumbar plexus, however, contributes to the nervous supply of these organs, inasmuch as the supra-pubic region, and the upper part of the scrotum or labium pudendi, receive branches from the ilio-hypogastric and ilio-inguinal nerves. The spermatic cord and cremaster or round ligament is supplied by the genital branch of the genito-crural nerve. Sympathetic filaments from the hypogastric plexus pass to the penis and prostate gland; while the testicle receives branches both from the hypogastric and spermatic plexuses. The vagina receives branches from the hypogastric plexus, and is also supplied by the fourth sacral and pudic nerves.

The external genitals may participate in neuralgia affecting various nerve regions, but isolated neuralgia of the genitals is sometimes met with. In the latter kind of cases several nerve regions are usually affected, so that it is difficult to determine the particular nerves implicated. The following varieties are met with:—

(1) *Neuralgia of the Penis and Mons Veneris*.—The pain has its seat in the glans, and extends to the root of the organ. The pain is increased by sexual intercourse, passing water, and may be accompanied by priapism and frequent ejaculations. The pain is violent, lancinating, and burning, and may be unilateral or bilateral.

(2) *Neuralgia Scrotalis (et labialis)* is a common symptom of lumbo-abdominal neuralgia. The scrotum or labium majus is often very tender to the touch.

(3) *Neuralgia Urethralis* is characterised by pain in the urethra during micturition, and increased desire to urinate. It is sometimes an early symptom of tabes dorsalis, but may arise from exposure to cold, or from morbid conditions of the urine.

(4) *Neuralgia Spermatica* is characterised by violent intermitting pain in the testis and epididymis, which radiates along the spermatic cord and down the thigh. In what has been described by Sir Astley Cooper as "irritable testis" the parts become extremely sensitive to pressure, and are liable to periodic enlargement. The attack is frequently accompanied by vomiting and general malaise. The affection chiefly occurs in young people, and is usually unilateral. Some regard this form of the disease as a variety of lumbo-abdominal neuralgia, but the opinion is now gaining ground that it is the sympathetic nerves which are chiefly implicated.

(5) *Neuralgia Ano-vesicalis* consists of morbid sensations in the region of the coccyx, associated with hyperæsthesia or anæsthesia of the skin of the perineal region, spasm of the sphincter ani and of the muscles of the urethra and bladder, and difficulty of micturition. It occurs in patients suffering from tabes dorsalis, and may be caused by onanism and excessive sexual indulgence.

*Treatment.*—The milder forms usually yield to the ordinary treatment for neuralgia, whilst the severer forms prove most intractable to all kinds of treatment. Narcotics, in the form of subcutaneous injection, and suppositories are useful and often necessary parts of the treatment. Specifics, such as arsenic, quinine, and oil of turpentine, often do good. Electric treatment has not yet been sufficiently tried, but the constant current should undoubtedly form part of the treatment. In spermatic neuralgia operative proceedings and even castration have been had recourse to.

### § 300. *Spasm in the Region supplied by the Lumbar and Sacral Nerves.*

(1) *Spastic contracture of the hip* was first described by



Stromeyer. It consists of a tonic spasm of the psoas and iliacus, of the quadratus lumborum, and occasionally of one or two of the muscles of the front of the thigh. The extremity is bent at the hip-joint, the tendon and muscular belly of the ilio-psoas muscle project strongly, the pelvis appears to be raised on the affected side, the limb is shortened, and in walking the patient inclines to that side. Any attempt to extend the limb causes acute pain in the tense muscles, and also in the knee. This affection is usually caused directly by disease of the lumbar vertebræ with psoas abscess, and in a reflex manner by disease of the hip-joint.

(2) Spasm of the *quadriceps extensor femoris* is rare. Tonic spasm of this muscle gives rise to rigid extension of the leg on the thigh, such as is observed in tetany and in neuralgia of the knee-joint. Clonic convulsion of the muscle was observed by Erb whenever the patella was touched in a case of articular hyperæsthesia.

(3) Contracture of the *adductors* of both thighs has been observed by Reitter caused probably by rheumatic inflammation of both hip-joints. It also occurs as a symptom of tetany.

FIG. 83.

FIG. 83. *Muscles of the Anterior Femoral Region (after Heath).*

- 1, Crest of the ilium.
- 2, Its anterosuperior spinous process.
- 3, Gluteus medius.
- 4, Tensor vaginae femoris; its insertion into the fascia lata is shown inferiorly.
- 5, Sartorius.
- 6, Rectus femoris.
- 7, Vastus externus.
- 8, Vastus internus.
- 9, Patella.
- 10, Iliacus internus.
- 11, Psoas magnus.
- 12, Pectineus.
- 13, Adductor longus.
- 14, Part of the adductor magnus.
- 15, Gracilis.

§ 301. *Paralysis of the Muscles supplied by the Lumbar and Sacral Nerves.*

(1) *Paralysis in the Region of Distribution of the Crural Nerve.*—The crural nerve supplies the iliacus, the quadriceps extensor femoris, the sartorius, and, in part, the pectineus.

Paralysis of this nerve arises from injuries of the vertebral column and pelvis, from tumours, and extravasations of blood

FIG. 84.



FIG. 84. *Deep Muscles of the Gluteal Region (after Heath.)*

1. Ilium.
2. Sacrum.
3. Posterior sacro-iliac ligaments.
4. Tuberosity of the ischium.
5. Great sacro-sciatic ligament.
6. Lesser sacro-sciatic ligament.
7. Trochanter major.
8. Gluteus minimus.
9. Pyriformis.
10. Gemellus superior.
11. Obturator internus, passing out of the lesser sacro-sciatic foramen.
12. Gemellus inferior.
13. Quadratus femoris.
14. Adductor magnus.
15. Vastus externus.
16. Biceps.
17. Gracilis.
18. Semi-tendinosus.

The tendon of the obturator externus should appear between the gemellus inferior and the quadratus femoris.

in the cauda equina; but it is rare as an isolated affection. It may succeed inflammation of the knee-joint, and also occurs in consequence of psoas abscess, in which case it is preceded by symptoms of irritation. It may be caused by fractures of the thigh and dislocation of the hip-joint, wounds and various other injuries of the lower abdominal and crural regions, neuritis, and by pelvic and crural tumours; it is a frequent symptom of all forms of spinal paralysis, and more rarely of cerebral paralysis.

*Symptoms.*—The patient is unable to flex the leg at the hip-joint, or to raise the body from the recumbent position, and the muscles are often atrophied. He cannot extend the leg, and when sitting, cannot move the leg forwards. Standing and sitting upright are rendered insecure; and walking, jumping, and running are rendered difficult or



impossible, and the difficulty is much increased when both crural nerves are affected.

Disturbances of sensibility are frequently observed, which extend over the lower two-thirds of the thigh, the region of the knee, and the inner side of the leg and foot.

(2) *Paralysis in the Region of Distribution of the Obturator Nerve.*—The obturator nerve supplies the adductors of the thigh, the gracilis, the obturator externus, and, in part, the pectineus. Paralysis of this nerve is rarer than crural paralysis, but it is frequently associated with the latter. Strangulated obturator hernia, and pressure of the head of the child or of obstetric instruments in difficult deliveries, may cause paralysis of the obturator nerve.

*Symptoms.*—The patient is incapable of adducting the thigh, of pressing the knees together, or of crossing one leg over the other. Rotation of the thigh outwards is rendered difficult, and the affected leg soon tires in walking. Some disturbances of sensibility, extending down the inner side of the thigh as low as the knee, are perceptible.

(3) *Paralysis of the Muscles supplied by the Gluteal Nerves.* The gluteal nerves supply the tensor fasciæ and the gluteus minimus and medius.

FIG. 85.

FIG. 85. *Muscles of the Posterior Femoral and Gluteal Region (after Heath).*

- 1, Gluteus medius.
- 2, Gluteus maximus.
- 3, Vastus externus covered in by fascia lata.
- 4, Long head of biceps.
- 5, Short head of biceps.
- 6, Semi-tendinosus.
- 7, Semi-membranosus.
- 8, Gracilis.
- 9, Part of the inner border of the adductor magnus.
- 10, Edge of sartorius.
- 11, Popliteal space.
- 12, Gastrocnemius; its two heads.

This form of paralysis is rare, occurring generally as a symptom of paralysis due to tumours and lesions of the cauda equina, to fractures of the sacrum and pelvis, and to spinal disease. The most prominent characters of pseudo-hypertrophic paralysis, and sometimes of progressive muscular atrophy, are not unfrequently due to paralysis and atrophy of the gluteal muscles.

*Symptoms.*—Rotation of the leg both inwards and outwards is interfered with, and the power of abduction is impaired. Some uncertainty is felt in standing, the patient experiences great difficulty in ascending stairs, and when the body is inclined forwards, it is difficult to raise it to the erect posture. The characteristic rotation of the pelvis (§ 30), produced by the contraction of the gluteus medius during locomotion, is not effected, and the muscle is not felt rigid on the side of the active leg, as in health, when the hand is laid over the pelvis above the trochanter. Atrophy of the muscles is common, but disturbances of sensibility are only present when other nerves are coincidentally affected.

*Treatment.*—The treatment of the various forms of spasm and paralysis in the region of distribution of the lumbar nerves must be conducted on general principles, and does not require to be described in detail.



## CHAPTER VIII.

## DISEASES OF THE SACRAL AND COCCYGEAL NERVES.

§ 302. *Neuralgia in the Region of the Sacral Plexus.*

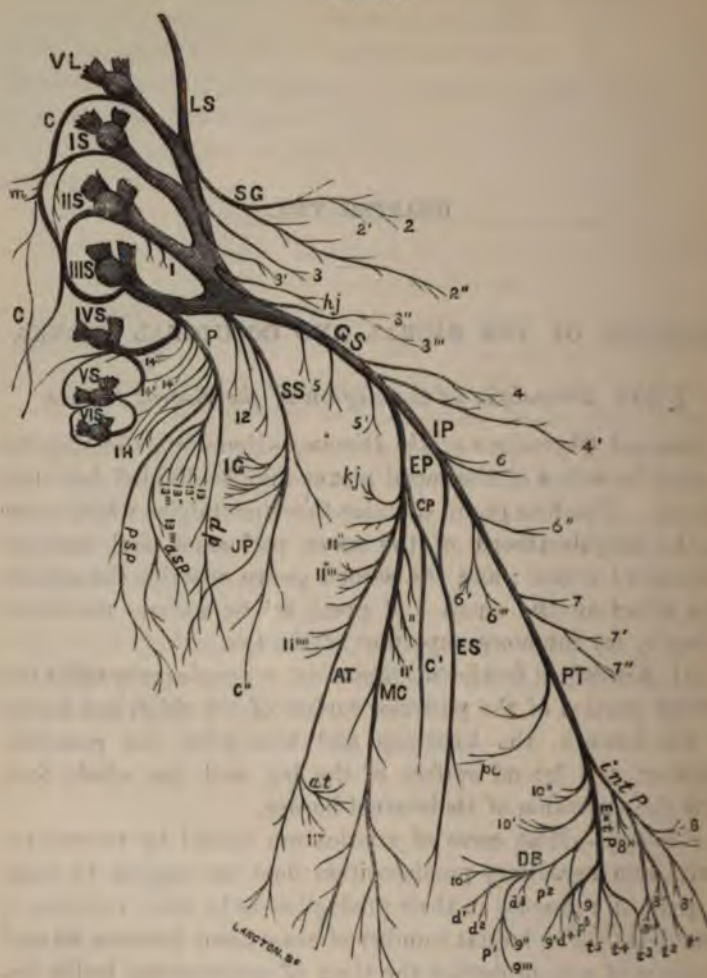
*General Characters of the Disease.*—Neuralgia affecting the sensory branches of the sacral plexus may be divided into two groups. The first group contains those neuralgias which occur in the neighbourhood of the anus, perinæum, and external generative organs; and the second group contains the neuralgias affecting the small and great sciatic nerves, the latter being by far the more important of the two.

(1) *Neuralgia Ischiadica (Sciatica).*—Sciatica may affect the greater portion of the posterior surface of the thigh and a part of the buttock, the knee-cap and knee-joint, the posterior, anterior, and lateral surface of the leg, and the whole foot, with the exception of its internal border.

*Etiology.*—Most cases of sciatica are caused by various injuries, and hereditary predisposition does not appear to exert so great an influence in their production as in other varieties of neuralgia. The largest number of cases occur between 40 and 50 years of age, or during the time of commencing bodily degeneration. After the age of 30 years the number of males affected greatly exceeds that of females.

The exciting causes are more potent in the production of the disease than the predisposing. Of these exposure to cold is probably the most frequent. Injuries to the nerve also commonly cause sciatica. Gunshot injuries sometimes occasion the severest and most intractable forms of sciatica, and falls on the buttock, prolonged and difficult labour especially when

FIG. 86.

FIG. 86. *Sacral and Coccygeal Nerves.*

VL, IS, IIS, IIIS, IVS, VS, VIS.—Fifth lumbar, and first, second, third, fourth, fifth, and sixth sacral nerves.

LS, Lumbo-sacral cord.

c, c', Posterior cutaneous nerves.

m, Branches to muscles of back.

1, Branches to piriformis muscle.

3, Muscular branches to obturator internus.

3', " " gemellus superior.

3'', " " gemellus inferior.

3''', " " quadratus femoris.

SG, Superior gluteal nerve.

2, Muscular branches to gluteus medius.

2', " " gluteus minimus.

2'', " " tensor vaginae femoris.



## SS, Small sciatic nerve.

12, Muscular branch to gluteus maximus.

I G, Inferior gluteal nerve (cutaneous).

[external labium.

I P, Inferior pudendal nerve, cutaneous to perinæum and scrotum, or

C', Cutaneous branch to back of thigh and upper part of leg.

## P, Pudic.

dp, Dorsalis penis seu clitoridis.

13, Muscular branch to transversus perinæi.

13', " " erector penis.

13'', " " compressor urethræ.

13''', " " accelerator urinæ.

13''', Branch to the bulb.

asp, Anterior superficial perinæal } to perinæum and scrotum,

psp, Posterior superficial perinæal } or external labium.

I H, Inferior hæmorrhoidal.

## IVS, Fourth sacral nerve.

14, Muscular branch to levator ani.

14', " " sphincter ani.

14'', " " coccygeus.

## GS, Great sciatic nerve.

hj, Branch to hip-joint.

4, Muscular branches to semi-tendinosus.

4', " " semi-membranosus.

5, " " adductor magnus.

5', " " biceps.

## IP, Internal popliteal.

6, Muscular branch to gastrocnemius (inner head).

6', " " " (outer head).

6'', " " popliteus.

6''', " " soleus.

## PT, Posterior tibial nerve.

7, Muscular branch to tibialis posticus.

7', " " flexor longus digitorum.

7'', " " flexor longus pollicis.

## pc, Plantar cutaneous.

## Int. P, Internal plantar nerve.

8, Muscular branch to abductor pollicis.

8', " " flexor brevis pollicis.

8'', " " first lumbricalis.

8''', " " second lumbricalis.

8x, " " flexor brevis digitorum.

t1 to t3, Digital branches.

## Ext. P, External plantar.

t4 and t5, Digital branch to fifth and outer half of fourth toe.

10', Muscular branch to flexor-accessorius muscle.

10'', " " abductor minimi digiti.

9, " " flexor minimi digiti.

9', " " fourth lumbricalis.

## DB, Deep branch of external plantar nerve.

9'', " " transversalis pedis.

9''', " " third lumbricalis.

p3, p2, p1, " { third, second, and first plantar interos-

sei muscles.

d4, d3, d2, d1, " { fourth, third, second, and first dorsal

interossei muscles.

10, " " adductor pollicis.

## ES, External or short saphenous to outer side of foot.

## EP, External Popliteal.

## CP, Communicans peronei.

## C', Cutaneous to outer side of leg.

## MC, Musculo-cutaneous nerve.

11, Muscular branch to peroneus longus.

11', " " peroneus brevis.

## AT, Anterior tibial nerve.

11'', Muscular branch to tibialis anticus.

11''', " " extensor longus digitorum.

11'', " " extensor longus pollicis.

11x, " " extensor brevis digitorum.

Ki, " " knee-joint.

at, Articular branch to tarsus.

the forceps is used, and fractures may be mentioned as causes of the disease.

Dr. Anstie regarded violent exertion of the lower extremities

FIG. 87.



FIG. 87. Second Stage of Dissection of Sole of Foot (from Hirschfeld and Leveillé).

- 1, Internal annular ligament.
- 2, Flexor brevis digitorum (cut).
- 3, External plantar nerve.
- 4, External plantar artery.
- 5, Internal plantar nerve.
- 6, Abductor minimi digiti.
- 7, Internal plantar artery.
- 8, Accessorius muscle.
- 9, Abductor pollicis.
- 10, Flexor longus digitorum.
- 11, Flexor longus pollicis.
- 12, Flexor brevis minimi digiti.
- 13, Digital branches of internal plantar nerve.
- 14, Digital branches of external plantar nerve.
- 15, Flexor brevis pollicis.
- 16, One of the lumbricales.

as a frequent cause of sciatica, especially at the period of commencing bodily degeneration. Mechanical pressure caused by sitting on hard seats, or by accumulation of feces in the sigmoid flexure, enlargements and displacements of the uterus, pregnancy, and every kind of tumour which presses upon the pelvic organs may induce severe sciatica. Hæmorrhoids, and conditions inducing congestion of the various plexuses of the pelvis, such as obstruction of the portal circulation and habitual constipation, are not unfrequent exciting causes of sciatica.

#### *Anatomical Changes.*—

Various changes have been found in the nerve itself in cases of intractable neuralgia. The changes most commonly described are congestion, œdema, neuritis, exudation, deposit of tubercle, and ossification of the neurilemma, tumours, and neuromata.

Diseases of the vertebræ and of the spinal cord, as spinal meningitis, myelitis, and locomotor ataxy, may cause sciatica, and it may follow typhoid fever, or result from syphilis.



*Symptoms.*—An attack of sciatica is generally preceded by premonitory symptoms, such as a sensation of fluid trickling over the skin, or a feeling of cold or heat, formication, and a sense of stiffness and dragging. After a time the symptoms of true neuralgia make their appearance. Lightning-like pains are felt, gradually increasing in intensity, and leading to a violent paroxysm. The pain proceeds from one or more fixed points, and usually radiates in a descending direction, but it occasionally radiates in an ascending, and sometimes in several directions. It is generally seated in the skin, but may be felt in and between the muscles, or even in the bones. There is generally an exacerbation of the pain at night; but it does not completely disappear during the day, and then there is a sense of tension and uneasiness in the affected limb. Occasionally, however, the pain is easier at night, and worse during the day.

Every movement of the limb generally augments the pain, and such trifling actions as are involved in turning in bed, coughing, sneezing, and straining at stool, or even simple contact of the bed-clothes, may bring on a paroxysm; hence the limb is maintained in a fixed position, with all the joints slightly bent. Patients are, however, occasionally met with who find relief in walking about, and who cannot remain in bed.

The area in which the pain is felt varies considerably in different cases. The pain is most commonly felt in the posterior surface of the thigh, commencing in the neighbourhood of the sciatic foramen, and extending to the popliteal space and calf of the leg. The next part which is most frequently affected is the region of distribution of the peronæus nerve, namely, the anterior and external surface of the leg, and dorsum of the foot. The region of distribution of the tibial nerve is more rarely affected, though cases have been observed where the pain was confined to the sole of the foot (neuralgia plantaris). One variety of the latter is so important as to deserve separate description.

Sometimes the whole area of distribution of the nerve is affected, and the violence of the pain may shift from one area to another. Violent pains in the sacrum and loins are usually felt along with sciatica, from implication of the posterior branches of the sacral nerves.

*Painful points* are rarely absent, although it is somewhat difficult to detect them in some cases. The places which are apt to be specially tender are : (1) A series of points representing the cutaneous emergence of the posterior branches, reaching from the lower end of the sacrum up to the crista ilii ; (2) A point opposite the emergence of the great and small sciatic nerves from the pelvis ; (3) A point opposite the cutaneous emergence of the ascending branches of the small sciatic, which run up towards the crista ilii ; (4) Several points at the posterior aspect of the thigh corresponding to the emergence of the cutaneous nerves ; (5) A *fibular point* at the head of the fibula corresponding to the division of the external popliteal ; (6) An *external malleolar point* behind the ankle ; (7) An *internal malleolar point*. The sacral plexus itself can be reached by examination *per anum* or *per vaginum*, and is frequently found painful on pressure.

The pain of sciatica may radiate to other nerve territories. It may shoot into the lumbar nerves and their branches, into the sciatic nerve of the opposite side, or into more remote nerve regions. Partial anæsthesia of portions of the skin of the affected extremity is frequently observed, and hyperæsthesia of circumscribed areas is not uncommon.

Motor disturbances are more frequent in sciatica than in any other form of neuralgia. Complete paralysis is rare, but in a large proportion of cases of long standing there is a considerable diminution of motor power, and consequently the gait is limping. The limp may occasionally be caused by the restriction of the movements of the leg on account of the pain, but as a rule it is the result of a paretic condition of the muscles.

Convulsive movements of the legs are met with in a considerable proportion of cases. Cramps of particular muscles may occur, consisting sometimes of slight fibrillary contractions, and at other times of strong convulsions. The cramps generally supervene at night when the patient is falling asleep.

The most usual vaso-motor and trophic disturbances are pallor and coldness, or redness and heat of the surface, increased secretion of sweat, atrophy, or occasionally hypertrophy of some of the muscles. In a case of sciatica which I saw recently, in consultation with my friend Mr. Stocks, the relative coldness of the affected limb was very well marked.

The sleeplessness caused by the constant pain may impair the appetite and lead to exhaustion, but sciatica of itself is never fatal.



FIG. 88.

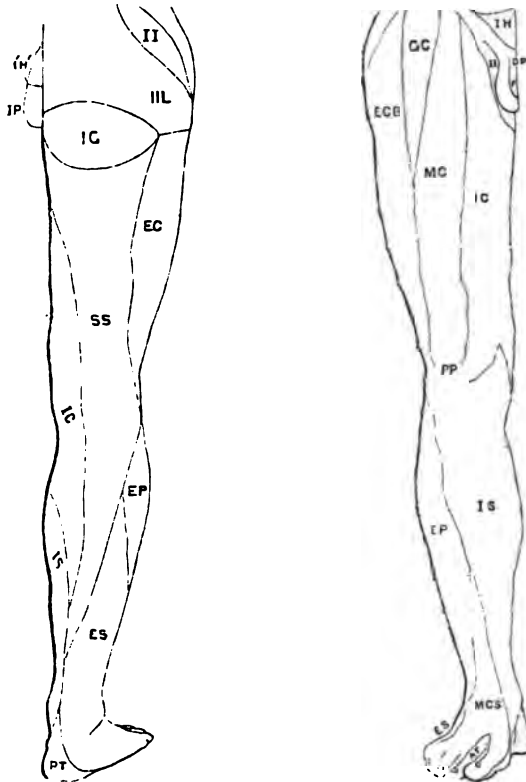


FIG. 88 (after Flower). *Cutaneous Nerves of the Lower Extremity. Anterior View of Lower Extremity.*

**Lumbar Plexus.**

- IH, Ilio-hypogastric nerve.
- II, Ilio-inguinal.
- IIL, Second lumbar nerve.
- GC, Genito-crural.
- EC, External cutaneous.
- MC, Middle cutaneous.
- IC, Internal cutaneous.
- IS, Internal saphenous.
- PP, Plexus patellæ.

**Sacral Plexus.**

- DP, Dorsalis penis of pudic.
- IP, Inferior hæmorrhoidal of pudic.
- P, Superficial perineal of pudic and inferior pudendal of small sciatic.
- IG, Inferior gluteal of small sciatic.
- SS, Small sciatic.
- EP, Branches from external popliteal.
- ES, External saphenous.
- MCS, Musculo-cutaneous.
- AT, Branches of anterior tibial.
- PT, Branch of posterior tibial.

The *electric sensibility* of the skin to the faradic current is slightly diminished when anæsthesia is present.

*Course, Duration, and Terminations.*—Sciatica, as a rule, lasts several weeks even in favourable cases. Occasionally recovery may take place in a few days, but more frequently it persists for months. The attacks supervene irregularly, each commences gradually, rises to a certain intensity, at which it remains with some variations for a longer or shorter time, and then gradually subsides. Improvement is usually very gradual, and even when the pain has disappeared the leg remains weak and stiff and easily tired, and the patient is liable to a relapse on slight provocation. In cases unassociated with organic disease the usual duration of the pain varies from two to eight weeks, but when it is the result of organic changes it may last for thirty years.

*Diagnosis.*—The diagnosis of sciatica is surrounded by considerable difficulties, especially in women, in fat persons, and in ignorant people, who cannot give a good description of their subjective sensations. It may be mistaken for myalgia of the thigh or leg, but the pain in the latter affection has a diffused localisation, so that the patient indicates its seat with his whole hand, whilst in neuralgia he points to it with his finger. The pain in myalgia is also increased by the performance of definite movements.

Sciatica may also be very readily confounded with hip-joint disease; and in slowly-developing cases of the latter disease, without fever or deformity, an error in diagnosis can only be avoided with the exercise of the greatest care. It is still more difficult to distinguish between hysterical coxalgia and sciatica. The absence or presence of pain when the head of the femur is pressed against the acetabulum, the age and the state of the general health, the elongation or shortening of the leg, the shape of the lower part of the back, the situation of the painful points, the paroxysmal character of the pain and its mode of distribution, the presence or absence of fever and inflammation, and the mode of carrying the leg, must all be taken into account in forming a diagnosis. Inflammation of the psoas muscle can be readily distinguished from sciatica.

It is important to determine whether the disease be seated in



the course of the nerves, the plexus, the roots, or in the central parts of the nervous system. The nerve is affected in its course when the pain is localised in particular branches, and when the concomitant motor and vaso-motor disturbances are well marked. The plexus is probably affected when the pain has a wide distribution, and when tenderness is produced on pressure being made from the rectum. When the pain extends to the posterior branches of the sacral plexus, is localised in the bones, and has a well-marked lancinating character, it is more likely to be seated in the roots or centres. In order to distinguish whether the roots or centres are affected, attention must be paid to the nature of the concomitant symptoms. It is of the greatest importance in treatment to have determined the cause of the sciatica.

*Prognosis.*—The prognosis is not so favourable in sciatica as in crural neuralgia; but in recent idiopathic or rheumatic cases the prognosis is always favourable. Slowly-developing cases, associated with anæsthesia, paralysis, and atrophy, are usually obstinate, and when the neuralgia is only a symptom of some incurable disease the prognosis is very grave. Long duration and old age add to the gravity of the prognosis.

(2) *Neuralgia Plantaris.*—Although the tibial nerve is rarely affected, yet one variety of neuralgia in the area of distribution of this nerve is so important as to deserve special mention. Dr. S. Weir Mitchell, who has done so much to advance our knowledge of nervous diseases, was the first to give an accurate description of this affection under the title of "Cases of rare vaso-motor disturbance in the leg." But inasmuch as the vaso-motor disturbances are preceded and accompanied throughout by severe paroxysms of pain, I see no reason why the affection should not be regarded as one of *plantar neuralgia*, or perhaps as a neuritis of the plantar nerves.

The disease occurs nearly always in the male sex, and comes on after some constitutional disease as a low fever, or after prolonged exertion; and in a case under my care the affection was the sequel of an attack of gonorrhœal rheumatism. It is, indeed, probable that the cases of obstinate pain in the sole of the foot described by Dr. Elliotson as following gonorrhœal rheumatism belong to this category. The pain is in the ball of the great toe, or in the heel, and often ex-

part of the sole, and may reach the dorsum of the foot and leg. It is generally limited to circumscribed areas of one or both soles, and does not extend beyond. It is felt at first towards night, and is relieved

FIG. 89.



by the night's rest; it is increased by walking, the erect posture, or even by allowing the foot to hang down. The pain is at first of an aching character, but after a time it becomes of a burning character, and is then aggravated by warmth, relieved by cold and the recumbent posture. The most characteristic symptom of the affection, however, is a flushing of the painful area coming on with exertion, or when the feet are allowed to hang down. "The foot," says Dr. Mitchell, "gets redder and redder, the veins stand out in a few minutes as if a ligature had been tied around the limb, and the arteries throb violently for a time, until at length the extremity

becomes of a dark purplish tint. In the worst cases, when the patient is at rest, the limbs are cold, and even pale." In aggravated cases the pain is so severe as to render walking all but impossible, and when walking is persisted in intense redness and swelling are occasioned. The patient sleeps with uncovered feet, and in the worst cases crawls on his hands and knees, or is carried about in order to avoid placing his feet on the ground. The disease is at times progressive, and in the later stages it is associated with evidences of spinal disease, such as girdle pains, partial paralysis, and atrophy of some of the muscles of the leg. In an interesting case recorded by Dr. Sturge there was considerable diminution of the faradic contractility in all the muscles of the limb most affected. In one of the cases observed by Dr. Mitchell the disease, which had at first been confined to the feet, extended at a later stage to the hands also. In the case observed by me there was a tender spot over the centre of the heel, the whole course of the external plantar nerve was very tender to pressure, and painful points were found between the heads of the metatarsal bones at the bifurcation of the branches of the plantar nerves for the digits. The feet were generally bathed in a sour-smelling sweat, and the skin of the sole had a sodden appearance, becoming somewhat glazed during the paroxysm of pain and redness. These cases are extremely intractable to treatment. The accompanying diagram, borrowed from Dr. Mitchell, is almost an exact representation of the distribution of the redness in the case under my care.

The disease which is most liable to be mistaken for plantar neuralgia is



Podynia—an affection peculiar to tailors—described by Dr. Gross. This affection consists of burning pain in some part of the sole, but there is no redness. It appears to depend upon subacute inflammation of the periosteum. A painful spot on the heel may be observed after syphilis, but careful examination will reveal a node on the os calcis.

(3) *Neuralgia of the Coccygeal Nerves (Coccygodynia)*.—The chief symptom of coccygodynia consists of pain in the region of the coccyx when the patient sits or walks, and often also during micturition and defecation, especially if there be much straining. Pressure on the coccyx with the finger induces pain. The affection is generally observed in women, in whom it occurs in consequence of injury to the coccyx from a fall, during labour, or from exposure to cold, and it may originate spontaneously. It is probably caused by irritation of the bone itself or of its fibrous investment, but occasionally it may be neuralgic, and is always of long duration.

*Treatment*.—In neuralgic cases the usual remedies must be adopted. In other cases operative proceedings are necessary. The usual operations are extirpation of the coccyx, or, better still, separating the bone by subcutaneous section with a tenotomy knife from all nerves connected with it. The latter operation has proved successful in several instances.

*Treatment of Sciatica*.—The first indication of treatment is to remove the cause of the disease. When the disease arises in a patient suffering from hæmorrhoids, venous stasis, and constipation, purgatives and saline waters, such as those of Kissingen and Marienbad, give good results. An enema containing turpentine has been strongly recommended in some of these cases. When the neuralgia results from external disease, operative procedure may be necessary, such as removal of tumours, coaptation of fractures, resection of cicatrices, and removal of foreign bodies; or, if there be traumatic neuritis, a pressure bandage may be applied to the whole extremity. When the disease is of central origin, the appropriate treatment for the particular lesion must be adopted. Recent rheumatic cases, which are by far the most common, should be treated by diaphoresis, the milder counter-irritants, the vapour or Turkish bath, and absolute rest; and in chronic cases the

iodide of potassium and the indifferent thermal baths prove serviceable.

Blisters are one of the most useful agents in the treatment of sciatica, and they are best applied in the form of flying blisters over the course of the painful nerve. Anstie recommended the application of the blister over the sacrum, so as to be directly opposite the posterior roots of the nerve; but it has been found much more generally useful in practice to apply the blister over the course of the painful nerve.

The actual cautery is an excellent remedy in the more serious cases, and the best mode of using it is to cauterise the skin superficially in transverse lines over the affected nerve. In two severe cases of sciatica which resisted the ordinary mild treatment I injected seven minims of a strong solution of nitrate of silver (grs. x to the 3j) subcutaneously. This method produced severe pain, and in one of the cases—an anæmic woman—it was followed by what appeared from the description to have been some kind of convulsion. In both instances the neuralgic pain disappeared in a few days, but I would certainly not advise this treatment except in very obstinate cases, and in moderately robust individuals.

Narcotics cannot be dispensed with in cases of even ordinary severity, and the subcutaneous injection of morphia over one or other of the painful points is the most generally useful method of administration. A small blister may be applied over the course of the painful nerve, and after the skin has been removed the surface should be dusted with morphia finely powdered, from one-sixth to one-third of a grain being applied every three or four hours. Atropine may be tried if morphia fail. Trousseau recommended an issue on the buttock to which two or three pilules are applied daily, each containing three-quarters of a grain of extract of opium and the same quantity of belladonna, made up with gum tragacanth. Anodyne liniments and embrocations, and narcotic clysters or suppositories afford valuable aid in the treatment of the disease.

Electricity gives good results in many cases. The faradic is not so generally useful as the constant current. The former may be applied in the form of the electric brush, or with moist electrodes. The constant current, however, is



much more generally useful, and it is equally successful in recent and in chronic cases of the disease which do not depend upon incurable organic disease. The descending current with stable electrodes is the most effectual method of applying it. The anode should be placed over the sciatic foramen or upon the sacrum, and the cathode upon the specially painful parts. Remak's plan may also be adopted. According to this method separate portions of the nerve, from six to eight inches in length, are successively brought under the influence of the current, beginning at the sacrum and passing down to the feet.

Another method employed by Remak, under the name of circular current, consists in the stable application of the anode upon the trunk of the nerve and upon the painful points. This method requires strong currents to be used, with broad electrodes, so that the current may pass deeply. In severe cases, Benedict has recommended that one electrode should be introduced into the rectum and the others placed on the sacrum, so that the current should be applied as directly as possible to the seat of the disease. Ciniselli recommended a single galvanic element—one zinc and one copper-plate connected by a wire—to be applied to the affected limb. Such plates may be adapted to any part of the skin, and may be worn for hours or for days together, and good results are sometimes obtained.

Various specific remedies have been from time to time recommended. Rectified oil of turpentine has been for a long time employed as a remedy. It is best taken in the form of gelatine capsules, each of which contains about fifteen grains, and three to twelve may be taken daily at meal times. Quinine has not been found very useful in sciatica. Iodide of potassium, either alone or in combination with guaiacum, is useful in some cases, more especially in rheumatic cases, and if there be a syphilitic taint it should be given in large doses.

Hot fomentations are frequently beneficial, or poultices may be used if preferred. The indifferent thermal and mud baths have for a long time enjoyed great repute in the treatment, and probably the greatest benefit is obtained by those means in chronic rheumatic cases. The most noted watering places for the cure of sciatica are Teplitz, Gastein, Wildbad, Wiesbaden,

and Baden-Baden. The cold-water treatment and sea-water baths have also been found serviceable.

In severe and desperate cases, and in these only, surgical operations are justifiable. Resections of the smaller sensory branches may be undertaken without fear; but it should be remembered that the paralysis which is produced by resection of a portion of the sciatic nerve, or of either of its two terminal branches, is generally incurable. Stretching of the sciatic nerve, according to the plan of Nussbaum, is a much more promising operation, since it not only relieves the pain, but the voluntary control over the movements of the limbs remains unaffected. Mr. Heath, of Manchester, reports a case of obstinate sciatica successfully treated by stretching of the nerve.

Rest in the recumbent position should always be insisted upon in the treatment of sciatica. Attention should also be paid to the regulation of the diet of the patient, and to the condition of the digestive canal and to the state of the health generally. The form of plantar neuralgia described by Dr. Mitchell has hitherto proved intractable to every kind of treatment. Stretching of the posterior tibial nerve deserves trial.

### § 303. *Spasm of the Muscles supplied by the Sciatic Nerve.*

(1) *Spasm of the flexors of the leg* (biceps femoris, semi-tendinosus, and semi-membranosus) occurs in the form of tonic contraction in hysterical patients, in disease of the spinal cord, and in disease of the knee-joint. The leg is kept strongly flexed upon the thigh, and the contraction is sometimes carried so far that the heel is brought into contact with the buttock.

(2) *Spasm of the anterior muscles of the leg*, or those supplied by the peroneal nerve, is of rare occurrence. Weir Mitchell has described a peculiar and painful contracture of the tibialis anticus, peroneus longus, and gastrocnemius, which occurs in young people after long standing, and leads to deformity of the feet. Duchenne has shown that spasm of the peroneus longus has a great influence in producing certain forms of club-foot. He has also drawn attention to the fact that the spasms are of two kinds: the first consisting of persistent contracture of the



muscle; the second, of functional spasm, which only occurs when the leg is brought into use.

(3) *Spasm of the muscles of the calf*, or of the muscles supplied by the tibial nerve, is frequent. The well-known "cramps," which occur in the calf from contraction of the gastrocnemius, belong to this variety. Contractures of the sural muscles produce "pes equinus," the heel being strongly elevated, the point of the foot depressed, and the toes flexed. Cramps in the calf are observed in sciatica, and in hyperæsthesia of the knee-joint.

(4) *Diffused spasm of the whole inferior extremity, or of both extremities together*, occurs occasionally in hysteria. Sometimes spasm of the leg may occur as a motor aura in epilepsy, and tonic and clonic spasms of the lower extremities are frequently met with in diseases of the spinal cord.

*Treatment.*—The treatment must first be directed to remove all the exciting causes of the disease. The direct treatment consists of the faradic and galvanic currents applied in the usual way, nervine tonics, and antispasmodics, along with tenotomy and orthopædic measures, according to the nature of the case.

### § 304. *Tetany (Tetanilla).*

Tetany consists of paroxysms of tonic contractions of certain groups of muscles, affecting for the most part the muscles of the forearm and hand, and sometimes extending to all the muscles of the upper and lower extremities, but seldom attacking those of the lower alone.

*Etiology.*—The disease frequently manifests itself from the fourth to the sixth year, and next to this, in liability to its occurrence, is the period of puberty and youth; the majority of cases in adults occur in those who are between sixteen and thirty years of age. The disposition to the disease is increased during pregnancy, in the puerperal state, and during lactation. No association appears to exist between the affection and the occupation of the patient.

Exposure to cold appears to be the most common exciting cause of the disease; hence many physicians have regarded it

as a rheumatic affection, and the swelling of the joints, which is frequently associated with it, indicates that it has a close affinity with rheumatism.

It is often observed as one of the sequelæ of typhoid fever, measles, cholera, Bright's disease, intermittent fever, exhausting diarrhœa, and various other diseases; and Moxon has directed attention to the similarity between the symptoms of tetany and those caused by ergotism. Puberty, pregnancy, and lactation favour the development of the disease; but whether these causes act by reflex irritation or in some other way is not known. Dentition and irritation of the alimentary canal, as that produced by worms and undigested food, are well-known causes of the disease, and undoubtedly act by reflex irritation. Tetany has also been known to follow various emotional disturbances.

*Symptoms.*—The spasmodic condition which is the main feature of the disease is usually preceded by various sensory disturbances, such as a sensation of creeping or dragging, formication, feelings of heat and cold, and sometimes well-marked pain in the forearm and hand. After a time slight contractions or feelings of stiffness occur in particular fingers, which are intensified when any object is grasped, and these are soon succeeded by the fully-developed spasm.

The spasmodic condition consists not of one, but of a series of distinct attacks. Each attack begins with a rigidity of the hand and fingers. The thumb is strongly adducted, the fingers are approximated, the second, third, and fourth fingers are strongly, while the first is only slightly, flexed, and all are firmly applied to the thumb. The hand is, indeed, drawn into a peculiar conical form, a position which may be obtained by a powerful faradic excitation of the ulnar nerve. The wrist is at the same time strongly flexed towards the ulnar side; and in other cases the fingers are closed and rigid, so that the hand resembles a paw.

Sometimes the region of distribution of the median nerve may be affected, in which case the thumb is turned inwards and tightly enclosed by the spasmodically-bent fingers. In rare cases the hands are violently flexed backwards, the forearms are semi-flexed, the upper arms are strongly adducted, and the



forearms are thus crossed upon the epigastrium. The affected muscles feel hard and are tightly stretched and painful, and although their tension undergoes considerable variation during the attack, they never become entirely relaxed. They offer considerable resistance to passive extension, and when force ceases to be applied they generally return to the position assumed in the spasmodic state.

The spasm frequently extends to the inferior extremities, forcing them to assume a rigid and extended position. The toes are strongly flexed, and the patient experiences great pain and hyperæsthesia of the muscles, and he is quite unable to walk or to perform any kind of work.

The muscles of the back and neck are only attacked in very severe cases; and those of the abdomen and diaphragm are still less frequently affected, whilst the muscles of facial expression, those of the tongue and of mastication, are almost never involved. When the diaphragm is affected the attack is of an alarming character, and may prove immediately fatal.

The spasm may last for a few minutes, or a quarter of an hour, or even for some hours, and its violence then gradually abates, leaving for some time a painful feeling of fatigue and a certain amount of rigidity and immobility of the muscles. After a shorter or longer interval a fresh attack comes on. As a rule the intervals are a few hours in duration, the attacks recurring several times in the day; but days or even weeks may intervene between them. In very severe cases the attacks may follow one another so rapidly that the patient only remains free from spasm for a few minutes at a time.

The affection consists of a greater or lesser number of these attacks, which may last for a period of a few days or weeks, but generally continue for several months. The patient feels well and able to work in the intervals, but occasionally a certain amount of weakness is felt in the muscles which have been affected with spasm. The attacks supervene without any obvious cause, but Trousseau has shown that if during the interval the larger arterial or nervous trunks of the upper extremities be compressed, a well-marked attack may be induced in the course of one or two minutes, which lasts as long as the pressure is maintained.

Well-marked concomitant symptoms are usually, although by no means always, present. In addition to the pain in the muscles, a tearing sensation is experienced in the course of the nerve trunks. The attacks are preceded and accompanied by creeping sensations, formication, and other abnormal feelings. Cutaneous anæsthesia has been observed in a few cases, and muscular anæsthesia has been reported in one or two instances. Redness and œdematous swelling of the skin around the joints, congestion of the head, headache, giddiness, and humming sounds in the ears are amongst the occasional symptoms mentioned. Disturbance of the general health is rare, although a certain amount of fever is present in severe cases. Free perspiration is of common occurrence during the acme of the attack. Respiration may be interfered with by spasm of the diaphragm and other respiratory muscles. The mental functions are almost always unimpaired.

§ 305. *Paralysis of the Muscles supplied by the Sciatic Nerve.*

*Etiology.*—From the exposed position of the nerve it is liable to injuries of various kinds, such as laceration, section, mechanical pressure from the growth of tumours, fractures of the vertebral column, tumours of the cauda equina or of the pelvis, in short, injury or compression of the nerve in any part of its course.

Rheumatic paralysis of the sciatic nerve, the result of a chill, is much less frequent. Paralysis from neuritis are more frequent, and a certain amount of paralysis is frequently left after severe sciatica, and in these cases it is probable that the neuralgia was the result of neuritis. Paralysis of the sciatic nerve may also occur after acute disease, and as a symptom of hysteria. This nerve is always implicated in the various forms of spinal and in many cases of cerebral paralysis. It is also involved in pseudo-hypertrophic paralysis, but is seldom affected in progressive muscular atrophy.

*Symptoms.*—The paralysis may affect the nerve as a whole, or either of its two branches, the peroneal and tibial nerves, or it may be limited to branches supplying particular muscles.

(1) If the *musculo-cutaneous nerve* be affected, the foot



cannot be flexed or abducted, and can only be incompletely adducted. It hangs down in a flaccid condition with the toes depressed, so that walking is seriously interfered with, inasmuch as the depressed toes are apt to trip the patient at every slight inequality of the ground. The necessary elevation is given to the passive foot in walking by flexion at the hip-joint, so that the point is the last part of the foot to be raised from the ground; and on planting it down, the outer border of the foot and the toes touch the ground first. This mode of progression is frequently observed in cases of infantile paralysis, and the gait is very characteristic. Secondary contractions of the muscles of the calf are apt to occur, and then the gait is rendered still more peculiar and characteristic.

The part played by the several muscles in producing these symptoms is as follows: Paralysis of the tibialis anticus limits the dorsal flexion and adduction of the foot, and the inner border and the point of the foot can no longer be raised, though these movements may be in part vicariously executed by the extensor digitorum communis, and the extensor longus pollicis. Paralysis of the extensor digitorum communis likewise diminishes the dorsal flexion of the foot and the abduction of the foot in the

FIG. 90.

FIG. 90. *Muscles of the Front of the Leg* (from Wilson).

- 1, Quadriceps extensor inserted into the patella.
- 2, Subcutaneous surface of the tibia.
- 3, Tibialis anticus.
- 4, Extensor longus digitorum.
- 5, Extensor proprius pollicis.
- 6, Peroneus tertius.
- 7, Peroneus longus.
- 8, Peroneus brevis.
- 9, 9, Borders of the soleus muscle.
- 10, Part of the inner belly of the gastrocnemius.
- 11, Extensor brevis digitorum; the tendon in front of the figure is that of the peroneus tertius; that behind it, the peroneus brevis.

flexed position, and renders extension of the basal phalanges of all the toes impossible.

Paralysis of the extensor longus pollicis diminishes dorsal flexion and abolishes the power of extending the great toe. Paralysis of the peroneus longus renders abduction of the foot in the extended position impossible, the arch of the foot becomes flattened, and the inner border no longer touches the

ground because the head of the first metatarsal bone is no longer drawn downwards. A peculiar kind of flat foot is produced, which has been carefully described by Duchenne. If the peroneus brevis be paralysed, pure abduction of the foot is rendered impossible, which can only be accomplished along with dorsal flexion by means of the extensor digitorum communis, or along with plantar flexion by means of the peroneus longus. Paralysis of the extensor digitorum communis brevis impairs the extension of the basal phalanges of the four last toes. These various paralysees may occur in an isolated manner, or they may be combined in various ways, and then the muscles affected can only be recognised by the most careful and prolonged investigation.

(2) If the *tibial nerve* be paralysed, all the muscles at the back of the leg are affected, and consequently extension of the foot as well as

Fig. 91.



FIG. 91. *Superficial Muscles of the Back of the Leg* (from Wilson).

- 1, Tendon of biceps.
- 2, Tendons of inner hamstrings.
- 3, Popliteal space.
- 4, Gastrocnemius.
- 5, 5, Soleus.
- 6, Tendo Achillis.
- 7, Tuberosity of os calcis.
- 8, Tendons of the peroneus longus and brevis.
- 9, Tendons of the tibialis posticus and flexor longus digitorum.



flexion and lateral movement of the toes is impossible. Paralysis of the gastrocnemius and soleus prevents the foot from being extended, and renders it impossible for the patient to stand upon the toes; and a hook-like position of the foot is produced, partly in consequence of the paralysis, and partly in consequence of secondary contraction of the muscles of the front of the leg. Paralysis of the *tibialis posticus* diminishes the power of adducting the foot, or of raising its inner border. Paralysis of the *flexor communis digitorum* renders flexion of the two distal phalanges of the toes impossible. Paralysis of the *flexor pollicis longus* renders flexion of the great toe incomplete. Paralysis of the adductor and abductor pollicis abolishes the power of moving the great toe laterally; while paralysis of the interossei renders flexion of the first, and extension of the two distal phalanges of the toes, as well as separation of the toes, impossible; and a peculiar claw-like position is thus produced just as in the hand. The first phalanx is abnormally extended, the second and third are strongly flexed, and the toes no longer touch the ground with their bulbous extremities. Some

FIG. 92.

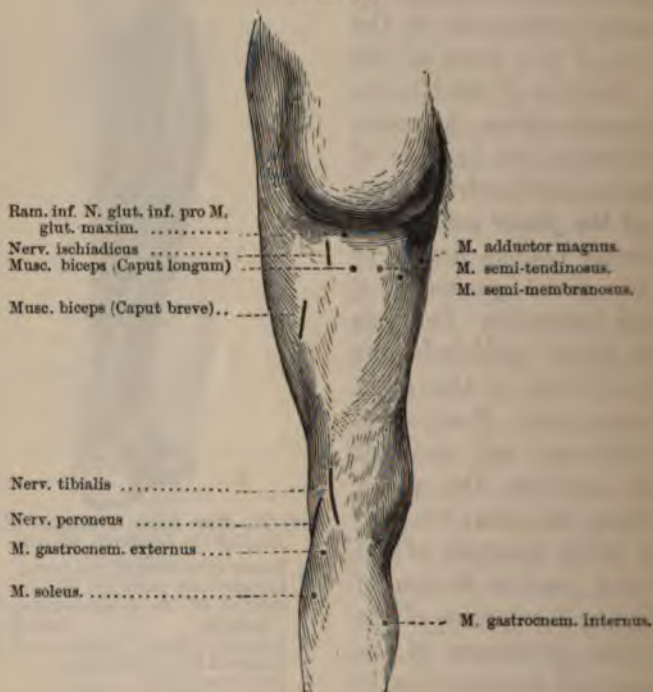
FIG. 93. *Deep Layer of Muscles of the Back of the Leg* (from Wilson).

- 1, Lower extremity of the femur.
- 2, Ligamentum posticum Winslowii
- 3, Tendon of the semi-membraneus muscle. [knee-joint.
- 4, Internal lateral ligament of the
- 5, External lateral ligament.
- 6, Popliteus muscle.
- 7, Flexor longus digitorum.
- 8, Tibialis posticus.
- 9, Flexor longus pollicis.
- 10, Peroneus longus.
- 11, Peroneus brevis.
- 12, Tendo Achillis divided near its insertion into the os calcis.
- 13, Tendons of the tibialis posticus and flexor longus digitorum, just as they are about to pass beneath the internal annular ligament of the ankle; the interval between the latter tendon and the tendon of the flexor longus pollicis is for the posterior tibial vessels and nerve.

pain and inconvenience are experienced after long standing or walking, but the functional disturbance produced is relatively small.

These paralyses of the muscles of the lower extremities cause various anomalies in the position of the foot and secondary alterations in the joints; but the details of these malformations must be referred to in surgical and orthopædic works.

FIG. 93.



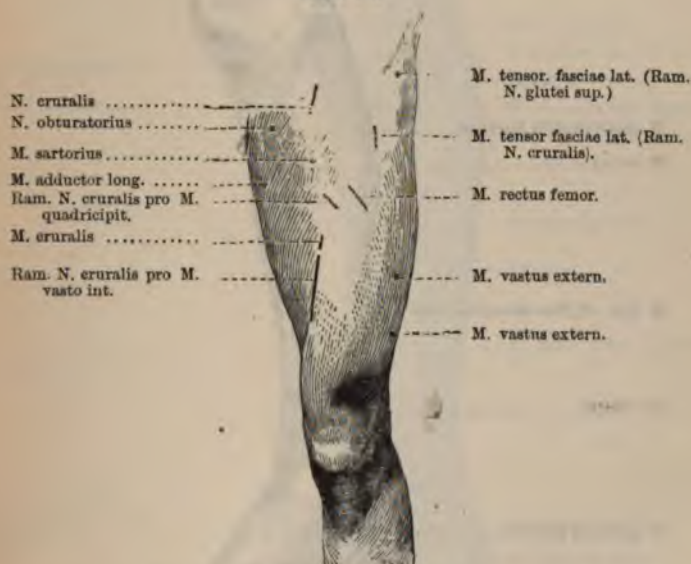
If the trunk of the sciatic nerve be affected, the branches distributed to the flexors of the leg, the semi-tendinosus, semi-membranosus, and biceps femoris are also paralysed, and the patients are unable to flex the leg upon the thigh, to approximate the heel to the gluteal region, or to offer any resistance when an attempt is made to extend the leg.

Paralyses of the sciatic nerve are usually accompanied by disturbances of sensibility, the extent of which depends upon



the cause of the paralysis. When the paralysis is limited to the peroneal region, the anæsthesia is limited to the anterior and external side of the leg, the dorsum of the foot, and the greater part of the toes. If the tibial nerve be affected, the posterior surface of the leg, the sole of the foot, and the plantar surface of the toes are the seats of the anæsthesia. The region of the knee, the back part of the thigh, and ultimately the buttock and perinæum are successively affected by anæsthesia, according as the lesion is situated at higher levels in the trunk

FIG. 94.



of the nerve. If the lesion be situated in the hollow of the sacrum or in the cauda equina, the anæsthesia affects the whole sacral region, the scrotum (or the labia) and penis, the urethra, bladder, and rectum.

*Vaso-motor* disturbances are usually present in the form of cyanosis, bluish-red marbled colouring, and coldness of the skin in the paralysed leg. Increase of temperature has occasionally been observed as a transitory symptom.

*Trophic disturbances* are not unfrequent in cases of severe peripheral paralysis of the sciatic. The most usual of these are

muscular atrophy, ulceration of the skin, eruptions of herpes and pemphigus, bed-sores on the sacrum, ankles, and heels.

*The diagnosis* between peripheral paralysis of the sciatic nerve and the paralysis resulting from central disease is not always easy. If the reflex actions are preserved in the paralysed muscles, the seat of the paralysis is central, although the converse does not hold true, since the reflex action is sometimes

FIG. 95.



abolished in spinal disease. The reaction of degeneration when associated with corresponding sensory disturbances is in favour of the peripheral origin of the disease; while, if it be not associated with any disturbance of sensibility, it is in favour of its spinal origin.

*The prognosis* is in all cases doubtful, depending both upon the nature of the cause and the accompanying trophic



disturbances, electrical reactions, and various other factors. In consequence of the great length of the sciatic nerve, the process of regeneration in the fibres requires a long time before it is completed, so that in traumatic and other forms of paralysis the affection is likely to be of long duration, and often proves incurable.

FIG. 96.



*Treatment.*—After removing the cause of the affection, where possible, the galvanic current should be employed along with a graduated system of gymnastics, and the use of thermal saline and mud baths. Operative measures are frequently necessary in order to remove deformities, but the reader must be referred for details to surgical treatises.





## PART II.—DISEASES OF THE SYMPATHETIC SYSTEM.

### CHAPTER I.

#### SUMMARY OF THE FUNCTIONS OF THE SYMPATHETIC SYSTEM.

THE sympathetic system of nerves consists of a vertebral and prevertebral portion; the *vertebral* portion is composed of a series of ganglia, united by a longitudinal cord (*Fig. 97, IC to C*); it descends along each side of the vertebral column from the head to the coccyx. The *prevertebral portion* consists of the numerous ganglia and plexuses of the head, chest, abdomen, and pelvis.

The sympathetic nerve communicates with the cerebro-spinal nerves immediately at their exit from the cranium and vertebral canal. It, however, unites with the fourth and sixth nerve in the cavernous sinus, with the olfactory in the nose, and with the auditory in the meatus auditorius internus. The branches of distribution accompany the arteries which supply the different organs, so that all the organs of the body are supplied by branches of the sympathetic.

A schematic representation of the vertebral portion of the sympathetic is given in *Fig. 97*.

#### § 306. *Functions of the Sympathetic System.*

1. *Reflex Action*.—The irritation is conveyed by afferent fibres to one of the ganglia of the sympathetic and then reflected through efferent fibres to unstriated muscular fibres, or to secretory cells, so that there are reflex secretory as well as reflex motor actions.

2. *Automatic Actions*.—Rhythmical discharges originating in the ganglia are conveyed by efferent fibres to plain muscular fibres or secretory cells, so that there are both motor and secretory automatic actions. It is pro-

bable, however, that many of the actions now regarded as automatic will prove to be reflex.

Automatic actions are under the regulation of inhibitory and stimulating or accelerating fibres.

### § 307. *Cervical Portion of the Sympathetic.*

1. *Vaso-motor fibres* for the corresponding half of the head. Claude Bernard showed that division of the cervical sympathetic in the lower animals produces a dilatation of the vessels of the head and neck on the side operated on, and elevation of temperature ranging from  $4^{\circ}$  to  $6^{\circ}$  C. Electrical excitation of the peripheral end of the divided cervical sympathetic contracts the dilated vessels of the head and neck, and lowers the temperature considerably below that of the opposite side.

2. *Oculo-pupillary Fibres*.—Division of the cervical sympathetic is also followed by contraction of the pupil (paralytic myosis), retraction of the globe of the eye, flattening of the cornea, and decrease in the size of the palpebral fissure, while irritation of the peripheral end of the divided sympathetic on the other hand produces mydriasis, prominence of the globe (exophthalmos), bulging of the cornea, and enlargement of the palpebral fissure. Both the vaso-motor and oculo-pupillary fibres have their origin, according to Claude Bernard, in the spinal cord, but the two sets do not issue at the same level. Section of the anterior roots of the two first dorsal nerves gives rise to the oculo-pupillary, but not to the vasculo-thermal phenomena; while on the other hand division of the ascending filaments of the thoracic sympathetic between the second and fourth ribs (in dogs) produces the latter without the former. Bernard consequently inferred that the centres of the oculo-pupillary and vaso-motor fibres exist at different levels in the cord. Budge first pointed out that the pupillary fibres issue from the spinal cord, in the region extending from the point of exit of the sixth cervical to that of the second dorsal nerve, and he consequently named this region the *centrum cilio-spinale inferius*. Budge believed that there was a second centre situated higher up in the cord, which was connected with the hypoglossal nerve by a communicating filament, and this he named the *centrum cilio-spinale superius*. Claude Bernard, on finding the influence of Budge's inferior centre on the pupil and eyeball, named it "*centrum oculo-pupillare*."

Contraction of the pupil after section of the cervical sympathetic is attributed to paralysis of the dilator pupillæ (paralytic myosis); while dilatation of the pupil (mydriasis), on irritation of the sympathetic, is attributed to contraction of this muscle. The exophthalmos, following irritation of the sympathetic, is usually referred to the action of the unstriated muscle of the orbit (*musculus orbitalis*), discovered by H. Müller, and situated in the neighbourhood of the inferior orbital fissure. This muscle, although imperfectly developed in man, is an important structure in rumi-



nants and receives its nerve supply from the spheno-palatine ganglion ; on contraction, it throws the globe of the eye forwards. Müller also discovered other unstriped muscles in the upper and lower eyelids of men, and the mammalia, which, by their contraction, take part in the enlargement of the palpebral fissure, and in the protrusion of the globe referred to.

After division of the cervical sympathetic, the eye is drawn inwards, caused by paresis of the external rectus, this muscle being supplied both by the abducens nerve and by filaments from the ascending branches of the superior cervical ganglion. Some authors think that the sympathetic exercises a *tonic influence on all the voluntary muscles of the eye*, and attribute several of the oculo-pupillary phenomena to the absence of this tonic influence when the sympathetic is divided.

3. *Trophic and Secretory Fibres.*—Irritation of the sympathetic is followed by contraction of the vessels of the salivary glands, and provokes a secretion which is rich in the specific elements of saliva. Ludwig has shown that when the discharge of this secretion is artificially impeded the pressure in the excretory duct of the gland may be greater than in the arteries supplying it ; hence it may be inferred that the cervical sympathetic contains special secretory branches for the salivary glands. After extirpation of the submaxillary ganglion a continuous secretion occurs, which may be increased by irritation of the organs of taste, but which speedily abates on the occurrence of structural alteration in the gland.

The nasal mucous membrane appears to be influenced in its nutrition by the sympathetic nerve. Vulpian found that irritation of the spheno-palatine ganglion was followed by increased secretion of the corresponding side of the nose.

The secretion of the lachrymal gland appears to be to some extent under the control of the sympathetic.

Brown-Séquard found a gradual atrophy of the eye on the side operated on in guinea-pigs and rabbits after section of the cervical sympathetic ; but Eulenburg and Guttmann did not find any trace of ophthalmia or atrophy of the eye in dogs one and a half months after the united vagus and sympathetic were cut. Brown-Séquard states that within a few months after division of the cervical sympathetic in guinea-pigs and rabbits he observed atrophy of the corresponding half of the brain. Vulpian subsequently obtained the same results.

4. *Cardiac Excito-Motor Branches.*—(a) Accelerating fibres for the heart which pass in the superior, middle, and inferior cardiac nerves of the sympathetic. The lowest cervical ganglion, as well as the highest thoracic ganglion, conducts accelerating fibres to the heart through the third branch of the ganglion. (b) The first and second branches are the roots of the depressor nerve.

5. Fibres proceeding to the cerebro-spinal organ which call into activity the cardiac inhibitory mechanism.

6. Fibres proceeding to the cerebro-spinal organ which stimulate the vaso-motor centre (pressor fibres).

FIG. 97.

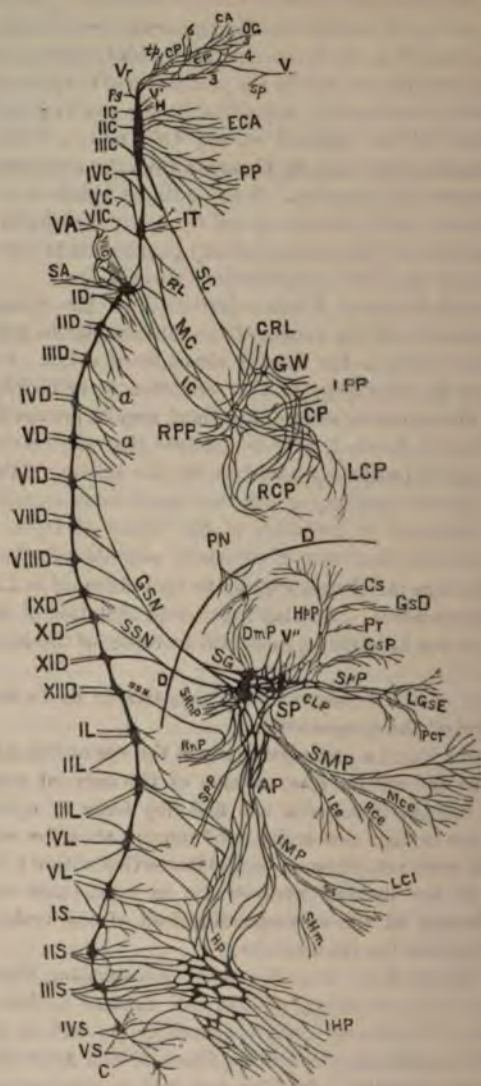


FIG 97 (Reduced from Flower). *Superior Cervical Ganglion of the Sympathetic: its connections and branches.*

IC to IVC, Branches of communication to four upper cervical nerves.

PS,	"	"	petrosal ganglion.
Vr,	"	"	ganglion of root of pneumogastric.
V,	"	"	ganglion of trunk of pneumogastric.
H,	"	"	hypoglossal nerve.
CP,	"	"	Carotid plexus.



- CP, Cavernous plexus.  
 CA, Branches accompanying internal carotid artery.  
 OG, " to ophthalmic ganglion.  
 th, To tympanic branch of glosso-pharyngeal.  
   3, to third nerve.  
   4, " fourth nerve.  
   5, " fifth nerve.  
   6, " sixth nerve.  
 V, Vidian nerve to spheno-palatine ganglion.  
 Sp, Large superficial petrosal from facial nerve.  
 EAC, Accompanying branches of external carotid artery.  
 PP, Pharyngeal plexus, formed by union with branches of vagus and glosso-pharyngeal nerves.  
 SG, Superior cardiac nerve.

*The Middle Cervical, or Thyroid Ganglion.*

- IVC to VIC, Branches of communication with fourth, fifth, and sixth cervical nerves.  
 IT, Inferior thyroid branches.  
 MC, Middle cardiac nerve.  
 RL, To recurrent laryngeal.

*The Inferior Cervical Ganglion.*

- VIIIC to VIIIC, Branches of communication with seventh and eighth cervical nerves.  
 IC, Inferior cardiac nerve.  
 CP, Cardiac plexus.  
 GW, Ganglion of Wrisberg.  
 LCP, Posterior, or left coronary plexus.  
 RCP, Anterior, or right coronary plexus.  
   CRL, Cardiac branches from pneumogastric or recurrent laryngeal nerves.  
   APP, To right anterior pulmonary plexus.  
   LPP, To left anterior pulmonary plexus.  
 ID to IID, Branches of communication from the first to the twelfth dorsal nerves.  
   a, a, To aorta, vertebræ, œsophagus, and posterior pulmonary plexus.  
 GSN, Great splanchnic nerve.  
 ssN, Small splanchnic nerve.  
 SSN, Smallest splanchnic nerve.  
 D, Diaphragm.  
 PN, Phrenic nerve.  
 SP, Epigastric, or solar plexus.  
   CLP, Coeliac plexus.  
   Cs, Cystic plexus.  
   GSD, Gastro-duodenal plexus.  
   C s P, Gastric or coronary plexus.  
   Py, Pyloric plexus.  
   SpP, Splenic plexus.  
   LGsE, Left gastro-epiploic plexus.  
   Per, Pancreatic plexus.  
   HpP, Hepatic plexus.  
   V<sup>o</sup>, Branches from pneumogastric.  
   DmP, Diaphragmatic plexus.  
   SG, Semilunar ganglion.  
   SRnP, Supra-renal plexus.  
   RnP, Renal plexus.  
   SpP, Spermatic plexus.  
 SMP, Superior mesenteric plexus.  
   Mce, Middle colic.  
   Rce, Right colic.  
   Ice, Ileo-colic.  
 AP, Aortic plexus.  
   IMP, Inferior mesenteric plexus.  
   LCI, Left colic plexus.  
   Sz, Sigmoid plexus.  
   SHm, Superior hæmorrhoidal plexus.  
 IL to VL, Branches of communication with the five lumbar nerves.  
 IS to VS, " " " " five sacral nerves.  
 C, " " " " coccygeal nerve.  
   HP, Hypogastric plexus. (pelvic viscera.  
   IHP, Pelvic, or inferior hypogastric branches to all the

§ 308. *Thoracic and Abdominal Parts of the Sympathetic System.*

The superior thoracic ganglion (*ganglion stellatum*) conducts accelerating fibres to the heart, which reach the ganglion by way of the cervical sympathetic cord and the root accompanying the vertebral artery (Von Bezold and Bever).

The cardiac plexus is constituted of fibres passing to and from the heart, and belonging to the vagus, depressor, and sympathetic nerves.

The splanchnic nerves contain the following fibres :—

1. Inhibitory fibres for the intestine. Irritation of the splanchnic in animals arrests the movements of the intestines.
2. Accelerating fibres for the intestine, surmised from the effect of post-mortem stimulation (Hermann).
3. Fibres inhibiting the renal secretion.
4. Vaso-motor fibres for the vascular regions of the abdomen.
5. Centripetal fibres, which inhibit the heart in a reflex manner, situated, in the frog, in the sympathetic cord (Bernstein).
6. Fibres, irritation of which causes the appearance of sugar in the urine.

Irritation of the plexuses of the sympathetic situated in the abdomen, as well as irritation of the cord, causes increased movements of the intestines, bladder, ureters, uterus, vesiculæ seminales, and spleen. Section or extirpation of the sympathetic cord and plexuses produces chiefly circulatory and nutritive disturbance. The supra-renal capsules are very rich in nerves, and contain in their interior cells resembling ganglion cells.



## CHAPTER II.

DISEASES OF THE CERVICAL PORTION OF THE  
SYMPATHETIC.

THE causes which give rise to functional disturbances of the cervical sympathetic are:—(I.) Compression by tumours; (II.) Injuries; (III.) Injury or disease of the cervical part of the spinal cord; (IV.) Functional disturbances to which no cause can be assigned.

## (I.)—COMPRESSION BY TUMOURS.

§ 309. *Compression of the Cervical Sympathetic Nerves by Tumours.*—A case is reported by Dr. Ogle in which the cervical sympathetic was compressed by a large carcinomatous growth in the neck, giving rise to contraction of the pupil on the affected side, and similar cases have been recorded by Heineke and Verneuil. In another case recorded by Dr. Ogle compression of the sympathetic on the right side was caused by a cicatrix, and on the side affected there was contraction of the pupil, flattening of the cornea, injection of the conjunctiva, congestion of the ear and neck, dilatation of the temporal artery, and elevation of temperature in the cavities of the mouth and nose. Willebrandt observed contraction of the pupil in cases of glandular swelling in the neck, which returned to its normal size when the tumours were reduced in size by the inunction of iodide of potassium. Compression of the sympathetic is not unfrequently caused by aneurism of the aorta, innominate, and carotid arteries (Gairdner, Coates).

Irritation of the cervical sympathetic may also be caused by

the pressure of tumours and other causes, and then dilatation of the pupil either alone or combined with pallor and decrease of temperature on the corresponding side results. In one of several cases published by Dr. Ogle the phenomena of irritation and those of paralysis alternated. The symptoms were caused by the formation of an acute abscess in the neck, and when the abscess was opened, the pupil, which was previously alternately contracted and dilated, gradually assumed its normal dimensions. On two subsequent occasions abscesses formed in the same region which were attended by similar symptoms. Eulenburg and Guttmann mention the case of a patient suffering from a vascular goitre limited to the right side. The symptoms were extreme mydriasis, complete immobility of the iris, considerable exophthalmos, and loss of the power of accommodation in the right eye, along with persistent lowering of the temperature of the auditory meatus of the same side. The vaso-motor are much more transient than the pupillary symptoms, and are frequently present only during the first few days or hours of the affection. Vulpian thinks that they are often overlooked.

#### (II.)—INJURIES.

§ 310. *Injuries to the Cervical Sympathetic.*—Weir Mitchell mentions the case of a soldier, who came under his observation ten weeks after a gunshot wound of the right side of the neck. The right pupil was unusually small, there was myopia of the right eye, slight ptosis, apparent sinking of the outer angle, decrease in the apparent size of the eyeball, and redness of the conjunctiva, and the right half of the face became much redder after violent exercise, which was followed by pain and flashes of light in the eye of the same side.

More or less similar cases have been recorded by Kämpf, Seeligmüller, and Bernhardt.

Seeligmüller also describes the case of a smith, who received a severe blow over the left supra-clavicular region, and two days later presented a great dilatation of the left pupil. The left palpebral fissure was wider than the right, and the left eyeball pushed forwards; while the left side of the head and neck was paler than the corresponding parts on the right.



## (III.)—INJURY OR DISEASE OF THE CERVICAL PART OF THE SPINAL CORD.

§ 311. *Injury to the Spinal Cord in the Neck, and to the Brachial Plexus.*—Hutchinson observed contraction of the pupil in injury of the spinal cord by a fracture of the seventh cervical vertebra. Rosenthal, on the other hand, found dilatation of both pupils, along with a remarkably small pulse (48 per minute) and paralysis of the four extremities in a man who had been stabbed in the neck in the neighbourhood of the sixth cervical vertebra. Out of a hundred cases of injury to the spinal cord in the neck, Rendu found only sixteen in which notice was taken of the state of the pupil. In some of these the irritative and in others the paralytic phenomena predominated.

Paralytic myosis is not uncommon in diseases of the spinal cord. Dr. Ogle observed contraction of the pupil in five cases of disease of the cervical portion.

Hutchinson observed myosis, immobility of the pupil, narrowing of the palpebral fissure, and a rise of temperature on the corresponding side of the face in a case of traumatic paralysis of the brachial plexus, and Seeligmüller subsequently confirmed this observation.

*Unilateral Hyperidrosis.*—Cases in which excessive sweating limited to one side of the head is associated with the other symptoms of paralysis of the sympathetic are rare, but unmistakable cases of the kind have been recorded by Chovstek, Pokroffsky, and Eulenburg and Guttmann. The case recorded by the last observers was that of a man 44 years of age, who perspired profusely on the left side of the face after moderate exercise. At the same time the left ear became red and its temperature raised, the conjunctiva on the same side was injected, and lachrymation was readily excited on that side. All these are paralytic phenomena, but it was remarkable that the left pupil was more dilated than the right, showing that the pupillary fibres were in a state of irritation.

## (IV.)—FUNCTIONAL AFFECTIONS OF THE CERVICAL PORTION OF THE SYMPATHETIC.

## 1.—CEPHALALGIA (HEADACHE).

§ 312. There is scarcely any disease, or symptom of disease, which assumes more Protean forms, and accompanies more varied conditions than headache. It is doubtful whether headache ought to be described under the diseases of the sympathetic

nerves ; indeed, it is certain that some forms of headache are not caused by disease of this system of nerves ; but, inasmuch as the vaso-motor phenomena give a decided character to one of the principal forms of the affection—hemicrania—it will be convenient to describe briefly all kinds of headache in this place. The various forms of trigeminal and occipital neuralgia are popularly included under the term headache, but these diseases must be excluded from any precise definition of the affection.

The following varieties of headache may be distinguished :—

(1) *Anæmic headache* is of a dull tensive character, usually affecting the temples, brow, and vertex, and extending along the sagittal suture. It is relieved by rest in bed, and the recumbent posture, and rendered worse by long maintenance of the erect posture. There is a disposition to faint, general pallor, palpitation, dizziness, and uterine disturbances in chlorotic females. All causes which exhaust the nervous system, as anxiety, night watching, and sexual excesses, aggravate this form of headache.

(2) *Hyperæmic headache* usually affects the whole head, the eyes are suffused, the carotids pulsate strongly, and the headache is accompanied by throbbing and sensations of pressure and weight in the head, agitation, hyperæsthesia, and illusions of the special senses. The headache is sometimes accompanied by intense redness and heat of the brow and vertex.

(3) *Hysterical headache* is met with in females, and is generally accompanied by other symptoms of hysteria. This form of headache is on the one hand closely allied to trigeminal neuralgia, and on the other to true migraine. The pain is sometimes diffused and deep-seated, but it is more frequently limited to one spot, and feels as if a nail were being driven through the skull ; hence it is called *clavus*. Hysterical headache is increased in severity during the menstrual period and by mental worry, whilst it is removed by amusement and anything which engages the attention.

(4) *Toxic headaches* are caused by various poisons circulating in the blood. One of the best examples of this form of headache is that which follows alcoholic intoxication. In the morning after a carouse a severe headache is experienced,



which is accompanied by a feeling of pressure and weight, chiefly localised in the deeper parts of the eyes and at the base of the brain. Severe headaches also follow the action of narcotics and anæsthetics. They are also caused by over-crowded rooms, and the inhalation of various gases, as carbonic oxide and sulphuretted hydrogen. Obstinate cephalalgia is one of the most common symptoms of uræmia.

(5) *Pyrexial Headache*.—Headache is also a frequent symptom of the acute infectious diseases, as scarlet and typhoid fevers, and of acute inflammatory diseases, as pneumonia. This form of headache is probably a variety of the congestive or hyperæmic headache, its main characteristic being that it is attended by a febrile temperature, while the other symptoms with which it is associated do not indicate that an intracranial disease is present. The headache is generally moderate in intensity, dull, deep-seated, increased by stooping, and accompanied by a feeling of lightness in the head, and not unfrequently by delirium.

(6) *Neurasthenic headache* occurs in those in which the nervous system is exhausted by mental anxiety and worry, night watching, and other depressing circumstances. General anæmia, combined with circumstances which depress and exhaust the nervous system, is the usual cause of this form of headache. The pain is generally deep-seated, heavy, dull, and oppressive, but varies considerably in its characters. It is often attended by a feeling of pressure and tension above the occiput, and is at times accompanied by great sensitiveness to touch in that region. The patient is generally compelled to suspend for a time all mental work.

(7) *Rheumatic headache* consists of a violent and tearing pain localised in the muscles of the head, or in the fascia of the occipitofrontalis muscle. It is often attended with marked tenderness of the scalp, and is usually brought on by exposure to cold.

(8) *Gouty headache* consists of frontal pain, and is generally attended by great depression of spirits, and not unfrequently by giddiness.

(9) *Sympathetic headache* may supervene on disease of almost all the peripheral organs, although it is most commonly associated with diseases of the digestive and sexual organs.

The most common form of this variety is the browache of gastric catarrh, but headache may accompany irritation of the intestinal canal or of the uterus and ovaries.

(10) *Syphilitic headache* is so important as to deserve special mention. An outburst of cerebral syphilis is generally preceded for many weeks by an intense and persistent headache. The pain is deep-seated and severe, and is either attended by a feeling of weight on the vertex or a sense of constriction as if the head were held fast in a vice. The patient often compares the pain to that which he imagines would be caused by successive blows struck on the head with a heavy mallet. The pain is sometimes distinctly circumscribed to a limited portion of the head; but, as a rule, it is more diffused, and may then occupy either the frontal, temporal, or occipital regions. It invades the whole head only on rare occasions. One of the chief characteristics of the headache is that it is constant, and never completely intermits, although it is liable to paroxysmal exacerbations of agonising intensity. Even in the slightest degrees of the affection the patient suffers greatly; he becomes sad, morose, excitable, and the mental faculties are so depressed as almost to incapacitate the patient for any work. In the severer forms of the affection the pain increases to the most agonising intensity, and the head becomes so sensitive to the touch that the patient is unable to lay it on a pillow. He sits up in bed grasping his head between his hands, groaning, or even screaming out. Syphilitic headache is often accompanied by an ephemeral delirium, and in the severest varieties the patient may become maniacal.

Another characteristic of the syphilitic headache is that it is liable to nocturnal exacerbations. The headache may be tolerable during the day, but it usually rises to such intensity at night that the patient is prevented from sleeping; so that those who suffer from syphilitic headache usually assert that they have not slept for weeks previously. The nocturnal exacerbations do not, however, always occur; and consequently an intense and persistent headache should of itself suggest the possibility of syphilis.

(11) *Organic Headache*.—The severe headache which accompanies structural disease within the cranium may be called organic headache (Day). Intense headache of a fixed character



is one of the most constant symptoms of intracranial tumours. Dr. Hughlings Jackson thinks that frontal headache is generally referrible to abdominal affections, vertical headache to cerebral disturbance, and occipital pains to disorders of the circulation, and more especially to anæmia. But the pain of cerebral disease may be frontal or occipital, or it may extend over the whole head, although it is more frequently deeply seated in one spot. The pain is persistent and continuous, but liable to paroxysmal exacerbations of great severity. It is often attended with tenderness of the scalp, and pain may be elicited on percussing the skull over the seat of the tumour.

Any disease of the brain, or of its membranes, may be associated with headache, and it is probable that the pain is more severe when the cortex of the brain and the membranes are affected than when the lesion is more deeply seated. It must, however, be remembered that a deeply-seated lesion, attended by increase of volume, such as a cerebral tumour, will also cause irritation of the cortex and membranes of the brain. Headache is also a constant symptom of inflammatory and ulcerative processes in the skull and adjoining tissues, such as catarrh of the frontal and sphenoidal sinuses, inflammatory affection of the scalp, the fascia of the occipito-frontalis muscle, and the pericranium. It is likewise an almost constant symptom of syphilitic diseases of the skull, caries of the petrous portion of the temporal bone, and certain lesions of the eye and ear.

Hemicrania is the next form of headache which I shall describe, but its importance is so great as to deserve separate and extended consideration.

#### HEMICRANIA

(MIGRAINE—SICK HEADACHE).

Hemicrania consists of spontaneous attacks of pain in the head occurring in paroxysms, and usually more marked on one side of the head, although it may reach to the opposite side.

§ 313. *Etiology*.—Like many other nervous affections hemicrania is hereditary, being often transmitted from parent to child. It very generally follows the female line, being usually transmitted from mother to daughter. The inheritance of

hemicrania is not, however, always direct; but a certain neurotic tendency is transmitted, of which headache becomes one of the manifestations. In families with tendencies to neuropathic diseases individual members suffer from migraine, while others are attacked with epilepsy, insanity, and other diseases of the same class. The severest and most intractable case of migraine that I have ever seen was in a lady whose mother was an epileptic. The female sex is affected with hemicrania in about the proportion of five to one of the male sex. The predisposition to neuralgias in general is greater among women than among men, but the disproportion between the liability of the sexes is not nearly so great in the other neuralgias as in hemicrania.

With respect to age, the period of youth is decidedly favourable to the development of the disease. The statement of Tissot, that a person who is not attacked with migraine before his twenty-fifth year will be exempt from the disease for the remainder of his life, may be accepted as a good practical maxim, although it is liable to considerable exceptions. When there is a strong hereditary predisposition to the disease it may appear during childhood. The age of puberty is especially favourable to its development, and the majority of cases of hemicrania make their appearance about this time.

The influence of various dyscrasias in the production of hemicrania is not readily detected. Patients suffering from anæmia, chlorosis, constitutional syphilis, or who have rheumatic or gouty constitutions, are liable to be attacked with hemicrania; but it is doubtful whether they are proportionately more frequently attacked than other persons. Hemicrania is also met with in those who are subject to hysteria; but this does not happen nearly so frequently as has been supposed. The reason that the association between hysteria and hemicrania has been supposed to be so frequent is that the "clavus" of hysterical patients has been mistaken for true migraine.

Individuals engaged in occupations demanding excessive mental activity doubtless suffer from hemicrania more frequently than those whose occupations lead them to exercise their muscular system; but, with this exception, hemicrania appears to occur with about equal frequency in all professions



and ranks of life. Of the exciting causes of hemicrania very little is known. It is, however, so frequently associated with gastric disturbance that it has been called sick-headache, but whether the digestive derangement be a cause or an effect of the headache is very difficult to determine. It is probable that disturbances in the circulation of the blood may be to a certain extent operative in the production of the disease, inasmuch as women during the time of menstruation are specially liable to be attacked by the disease, and it not unfrequently disappears after the climacteric period.

§ 314. *Symptoms.*—Hemicrania consists of paroxysms of headache separated by intervals of shorter or longer duration, which are usually free from symptoms. The attack is frequently preceded by premonitory symptoms. From twelve to twenty-four hours before the attack the patient feels depressed and weary, along with a sense of pressure in the head and indisposition to continued work. The patient feels a chill and nausea, and may have attacks of yawning or sneezing, and not unfrequently he complains of *muscæ volitantes* and *tinnitus aurium*.

The headache of migraine is often preceded by an attack of scintillating scotoma (§ 204). In the case of a young girl, kindly sent to me by Dr. Sinclair a few days ago, the headache was preceded by transitory hemiopia, but the dark clouds which came over one-half of the fields of vision were not surrounded by coloured spectra. Each attack of hemiopia lasted for about half an hour, and was soon followed by severe hemicranial headache. The attack of scintillating scotoma is often accompanied by other interesting symptoms. The most usual of these are transitory impairment of cutaneous sensibility, along with tingling, numbness, and formication. There may also be deafness, loss of taste, embarrassment of speech, momentary incoherence, transitory paresis of one of the limbs, vertigo, and nausea.

The characteristic pain comes on by degrees in the course of the day, and almost never with the lightning rapidity of the pain of neuralgia. The patient generally wakes with the pain in the morning, and in these cases he has not unfrequently experienced great drowsiness the previous evening. The pain is confined to one-half of the cranium, but it is not generally strictly

so limited. It may begin as a dull pain over the forehead, and, as it increases in severity, it passes down to one eye, and remains fixed over the temple. Occasionally it is seated at the top or back of the head. The left side is more frequently attacked than the right, the frequency being, according to Eulenburg, in the proportion of two to one. The attacks occur by turns on each side of the head in some individuals, but in these cases one side of the head is attacked oftener and more severely than the other. Eulenburg gives the name of *hemicrania alternans* to these cases, and he thinks that this variety is specially liable to be associated with vaso-motor disturbances.

The pain is sometimes so violent as to deserve the name of neuralgia; but, instead of being tearing or darting, as in true neuralgia, patients describe it as being dull, burning, or bursting, and it is frequently associated with an intense feeling of sickness. With every beat of the heart the patient feels a throb of pain in the head; and the slightest movement which excites the circulation, even raising the head from the pillow, or the exertion of talking, augments the throbbing pain, so that it becomes almost unendurable.

There is complete anorexia, the patient being usually unable to swallow any food for twenty-four hours; but this rule is not without its exceptions, since some patients eat as usual notwithstanding the headache. Nausea is almost constantly present, and vomiting is a frequent accompaniment of the disease. The patient may also suffer from indistinct vision, hemiopia, or *muscæ volitantes*, and *tinnitus aurium*; there is a bitter taste in the mouth, which is usually attributed to concomitant derangement of the stomach or liver, but which is much more likely to be due to functional disturbance of the nerves of taste.

*Painful points* are absent in true hemicrania, although sometimes a spot above the *tuber parietale* is tender on pressure. Cutaneous hyperalgesia is not an uncommon symptom during an attack of migraine; the greater part of the forehead, temples, and parietal regions being in many cases very sensitive to light touches, while deep diffused pressure affords relief.

Deep pressure causes decided pain when applied over the region of the superior or middle cervical ganglion of the sympathetic; and at times when applied to the spinous processes of



the lowest cervical and uppermost dorsal vertebræ—the cilio-spinal region of the cord (Eulenburg).

It has also been shown, by O. Berger,\* that a morbid acuteness of the sense of touch (*hyperpselaphesia*) may be present. He found that the diameter of the circle of perception was only one line in the frontal region of the right or affected side; while it was four lines at the corresponding point in the opposite or unaffected side. Variations of temperature of  $0.4^{\circ}\text{C}$  were perceived on the right side, of  $0.8^{\circ}\text{C}$  on the left; and the electro-cutaneous test indicated increased sensibility on the affected side.

§ 315. *Varieties*.—The vaso-motor disturbances during the paroxysms of hemicrania are so marked that they give a decided character to the attack. Three varieties of these may be distinguished. The first form is mainly characterised by vascular contraction, hence the attacks in which this variety occurs are called *hemicrania spastica* or *sympathico-tonica*; the second is characterised by vascular dilatation, and the attacks in which this variety occurs as a concomitant symptom Eulenburg proposes to call *hemicrania angio-paralytica* or *neuro-paralytica*; and the third variety is a *mixed form*, in which the symptoms of the sympathico-tonic and neuro-paralytic varieties alternate. There are cases of migraine which present no appreciable local vaso-motor disturbances.

1. *Hemicrania spastica* or *Sympathico-tonica*.—At the height of the attack the face is pale on the affected side, the eye is sunk, the pupil dilated, and the temporal artery feels like a hard cord. The ear on the affected side is pale and colder than the other, the difference in temperature between the two being, according to Eulenburg, from  $0.4^{\circ}$  to  $0.6^{\circ}$  taken in the external meatus. Compression of the carotid on the affected side augments, and on that of the unaffected side diminishes the pain. The salivary secretion becomes very viscid, and it is greatly increased in quantity. Berger observed over two pounds discharged in a single attack. The pain is aggravated by every circumstance which excites the circulation and increases the arterial tension. Towards the end of the attack the affected side of the face and the ear becomes reddened, and this is accompanied by a sensation of heat and a rise in temperature, redness of the conjunctiva, lachrymation, and sometimes contraction of the pupil. A general feeling of warmth is felt, the heart palpitates, there is vomiting, an abundant discharge of watery urine, and sometimes even a watery discharge from the bowels.

2. *Hemicrania Angio-paralytica* or *Neuro-paralytica*.—At the height of the attack the affected side of the face is red, hot, and turgid, the conjunctiva is injected, the pupil is contracted, and there is an increased

\* Virchow's Archiv, liz., Heft 3 and 4, p. 324.

secretion of tears. At times there is narrowing of the palpebral fissure, retraction of the globe, and a falling of the upper lid, along with difficulty in performing its movements. The ear on the affected side is red and its temperature from  $0.2^{\circ}$  to  $0.4^{\circ}$  C above that of the opposite ear. The temporal artery is enlarged and beats with unusual force. Compression of the carotid on the affected side eases, and of that of the opposite side aggravates the pain. Sometimes the radial artery is small and contracted, and the pulse slow, beating from 48 to 56 times a minute, but these symptoms are not always present. Towards the end of the attack the affected side of the face becomes paler, and the other phenomena gradually pass off.

3. *The mixed form* of the affection requires no special mention. In a case observed by Berger, the neuro-paralytic attacks ran a much milder course than the spastic attacks, and especially with less vomiting.

§ 316. *Course, Duration, and Terminations.*—The duration of the attack of migraine is very variable. It usually lasts from a few hours to half a day, but continues at times a whole day, or several days, with remissions and exacerbations. If the pain be present on waking, it gradually wears off towards evening; the patient feels exhausted and falls into a sleep, from which he awakes generally free from pain. If it should come on during the day, it gradually increases in severity, and sleep is rendered impossible. It is sometimes aggravated by the recumbent position, the sitting or upright posture being the only one in which the patient can endure his sufferings. The attacks not unfrequently recur with great regularity at intervals of three or four weeks. In the female sex the attacks are often coincident with the catamenia, but this is by no means always the case. The intervals are as a rule free from pain, if we except a slight tenderness over the region of the superior cervical ganglion and the spinous processes of the superior cervical vertebræ. The attacks may be aggravated as well as induced by bodily and mental exertion, emotional disturbances, exposure to draughts of cold air, changes of temperature, and gastric disturbances. The attacks not unfrequently cease after the climacteric period in women, and after the fiftieth year of age in men. The symptoms may occasionally disappear either spontaneously or under the influence of remedies in young persons who do not manifest a hereditary tendency to the affection.

*Diagnosis.*—In forming a diagnosis of the nature of a headache, careful examination should be directed to the



external tissues of the head and to the functions of the brain, peripheral nerves, and organs of special sense. A careful examination of the condition of the digestive organs, the heart, and blood-vessels often affords valuable information with respect to the cause of a headache, while a chemical examination of the urine should never be neglected. In obstinate headaches lasting for years an ophthalmoscopic examination should always be made, as the condition of the fundus of the eye may indicate the existence of serious cerebral disease, as tumour, or uræmic or other poisoning. The circulatory and digestive organs should be carefully examined, and the existence or absence of hereditary predisposition or toxic influences ascertained. Valuable information may sometimes be obtained by percussing the skull.

The prognosis varies according to the nature of the cause of the headache, and except in the case of organic headache it is favourable as far as life is concerned.

§ 317. *Morbid Anatomy and Physiology.*—Inasmuch as hemicrania is not a fatal disease, no aid need be expected from morbid anatomy in the elucidation of its pathology. Pain is the predominant symptom of migraine to the patient, but the most significant symptoms to the physician are those connected with the vaso-motor disturbances, and the oculo-pupillary symptoms.

*In hemicrania sympathico-tonica* there is unilateral tonic spasm of the vessels of the head, such as may be caused experimentally by irritation of the cervical sympathetic or of the corresponding half of the cilio-spinal region of the cord. The other symptoms present, such as increase of the salivary secretion and dilatation of the pupil (spastic mydriasis), also indicate irritation of the cervical sympathetic, and this supposition is much strengthened by the fact that the region of the superior and middle cervical ganglia of the sympathetic and the spinous processes of the lowest cervical and uppermost dorsal vertebræ are tender to pressure during the attack, and sometimes even during the intervals of pain (Eulenburg). The symptoms of hemicrania angio-paralytica are characterised by an opposite condition of the vessels to that which is present in the spastic variety, due no doubt to a diminished action of the cervical

sympathetic and of the cilio-spinal centre of the affected side. The concomitant oculo-pupillary symptoms, such as contraction of the pupil (paralytic myosis), narrowing of the palpebral fissure, retraction of the globe of the eye, occasional ptosis, strengthen this conclusion, inasmuch as they are the well-known effects produced by section of the cervical sympathetic in animals.

Landois believes that the retardation of the pulse present during the attack depends upon direct irritation of the medulla and vagi. Various other symptoms show that the vaso-motor centre is in a state of irritation. The icy coldness of the hands and feet, the chilly sensations felt over the whole surface of the body, the suppression of perspiration during the attack, with the exception sometimes of the affected side of the head, the contracted state of the radial artery, and the increase of the arterial tension, may be explained by irritation of the vaso-motor centre. The irritation is followed by a corresponding degree of exhaustion, and then the contraction of the peripheral arteries is followed by relaxation; during the latter stage of the attack there is an increased secretion of saliva and urine, and watery stools may be passed.

With regard to the locality of the pain in hemicrania, many authors regard it as a variety of supraorbital neuralgia; but the symptoms of the two affections differ from each other in so many ways that they can hardly be regarded as belonging to the same category. Romberg thought that the pain in hemicrania was due to hyperæsthesia of the brain, and consequently he called it "neuralgia cerebialis," in order to distinguish it from peripheral neuralgia; and although this view is not accepted in its entirety, the opinion is gaining ground that the seat of the pain is within and not without the cavity of the cranium. All three divisions of the trigeminus send branches to the dura mater. The first division gives off the nervus tentorii of Arnold, which passes through the tentorium to the sinuses; the second division the branch which runs with the middle meningeal artery; and the third division the nervus spinosus of Luschka (*Fig. 98*).

Numerous nerves are found in the pia mater, in the form of plexuses around the vessels, and some of these extend into the



cortex of the brain. These nerves originate partly from the vertebral and carotid plexuses of the sympathetic, and partly from the cerebral nerves, especially the trigeminus, at their points of exit from the cranium. Very little is known with regard to the nerves of the arachnoid. It is supposed by some that the seat of pain in hemicrania is to be referred to the intracranial and meningeal branches of the trigeminus and the other nerves which accompany the blood-vessels, and not, as Romberg had supposed, to the cerebral mass itself.

FIG. 98.

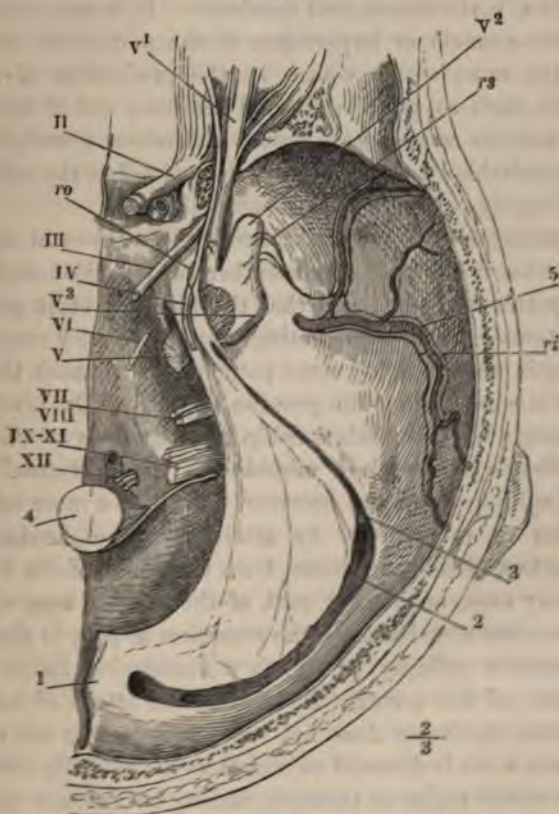


FIG. 98 (From Henle's Anatomie). *View of the Base of the Skull, showing the places of Exit of the Cranial Nerves, and the Sensory Nerves to the Dura Mater.*—The Roman letters indicate the Corresponding Cranial Nerves; ro, Nervus Tentorii of Arnold; r², Branch from the Second Division of the Fifth Nerve accompanying the Middle Meningeal Artery; r¹, the Nervus Spinosus of Luschka derived from the Third Division of the Nerve.

Du Bois-Reymond thought the pain was produced by irritation of the vascular nerves, due to a tonic spasm of the muscular coat of the vessels, and being similar to the pain felt in cramp of the calf or in tetanus, and in the smooth muscles of the uterus during labour. This supposition would account for the fact that the pain increases with the rise in arterial tension, and that each throb of pain corresponds to each pulsation of the temporal artery. Others believe that the temporary anæmia or hyperæmia of one-half of the head acts as a source of irritation to some or all of the various sensory nerves of the head, those of the skin, pericranium, and meninges. It is now recognised that either anæmia or hyperæmia of the peripheral nerves is one of the most active causes in the production of various neuralgias, such as prosopalgia and sciatica; and if variations in the vascular supply can induce cutaneous neuralgia, it is thought probable that a similar result may follow the variations of blood supply to the head in hemicrania.

The paroxysmal character of hemicrania, taken along with the fact that it occurs in the members of families manifesting a neuropathic tendency, and that the attack is often preceded by symptoms such as scintillating scotoma, closely resembling an epileptic aura, has led some pathologists to think that the affection is closely allied to genuine epilepsy. Dr. Liveing insists strongly upon the relationship between the two affections, and attributes the attack of migraine to "nerve storms."

Dr. Hughlings Jackson, however, has given a more scientific expression to this theory by attributing the headache of migraine to a discharging lesion from the cortex of the brain in the sensory area, or in that part of the sensory area which is the anatomical correlative of the sensation of pain in the head; while genuine epilepsy is due to a discharging lesion of the motor area of the cortex. But during the attack of migraine the nervous discharge does not remain limited to the sensory area, since some is directed outwards to the medulla oblongata and cilio-spinal region of the cord, causing irritation or paralysis of some of these centres, and giving rise to the vaso-motor and oculo-pupillary phenomena present during the attack.

§ 318. *Treatment.*—The treatment of headache may be



directed against either the cause of the disease, or the pain itself as a local symptom. In order to fulfil the first indication, the remedies for anæmia, hysteria, syphilis, nervous exhaustion, and for allaying local irritations, must be employed according to the nature of the case.

It is scarcely possible to adopt a causal treatment in the case of migraine, inasmuch as so little is known of the circumstances which concur to induce an attack. The ordinary aperient medicines which are generally prescribed for the cure of a headache are useless in migraine; but Dr. Wilks thinks that the act of vomiting occasionally affords relief. The vomiting, however, does not appear to act by removing indigestible substances from the stomach. The effect seems to be produced through the nervous system; hence this remedy cannot be regarded as one directed against the cause of the disease. The direct treatment may be subdivided into that which is appropriate during the intervals in order to prevent the attacks, and that which should be adopted with the view of removing or palliating the attacks themselves. Although the following remarks are particularly applicable to the treatment of hemicrania, yet the methods described are useful in other forms of headache.

The preparations of iron, especially the carbonate, have been strongly advocated by Hutchinson, Stokes, and others, and of their utility in the case of patients who are of weakly and anæmic constitution there can be no doubt. The tolerable regularity with which the attack recurs has led to the employment of the so-called antiperiodic remedies in the treatment of hemicrania. The remedies of this kind which have been employed are quinine, quinoidin, salicin, and arsenic, but it is very doubtful whether the recurrence of the attacks is prevented, or the paroxysms are rendered milder. Strychnia, nitrate of silver, sulphate of nickel, bromide of potassium, chloride of ammonium, oil of turpentine, and lupulin are other remedies which have been employed, but with doubtful success, although both the bromide of potassium and chloride of ammonium may be found of use during the attack. Chalybeate springs, mud and sea baths have each been found useful in the treatment of hemicrania, and the same may be said of the

use of hydropathy, and of residence in lofty mountain regions; but all these measures appear to influence the disease by improving the general health.

In the treatment of the attack every source of external irritation should be removed, the room should be moderately darkened, and all noises should be prevented. In the anæmic form the patient should lie flat on the back with the head a little raised, but in the hyperæmic form the patient usually prefers to maintain a sitting posture, and not unfrequently it is more grateful to rest the head against a hard substance. When I was a boy, an old servant in our family suffered from severe periodical headache, which prevented her from sleeping, and which, so far as I can remember, had all the characters of true migraine. Her remedy was to place a stone flag on her pillow, and by resting her head on it the combined hardness and coldness of the stone seemed to enable her to sleep, and next morning she woke without headache. Firm compression of the head by a handkerchief bound round it is an old remedy, and appears to give temporary relief. The application of cold may be of use. It may be applied in the form of a wet cloth bound round the temples, an evaporating lotion, or an ice-bag, the latter of which is the most effectual. Compression of the carotid in the neck will suspend the throbbing pain for a short time, but the effect is very transient, inasmuch as the blood finds its way to the brain through other channels.

Although quinine when given as an antiperiodic does not appear to exert much influence in preventing the paroxysms of pain, yet a dose of from five to fifteen grains given once or oftener may arrest an attack. The effect appears to depend upon an action on the vaso-motor nerves, since it has been proved experimentally that quinine in large doses contracts the arterioles and raises the arterial tension.

Ergot of rye, given in half drachm doses of the liquid extract, has also been found exceedingly useful in arresting an attack of migraine; and probably acts on the principle of contracting the blood-vessels. This remedy is therefore adapted for the treatment of the angio-paralytic variety.

Dr. Ringer speaks highly of *cannabis indica*, and considers it one of the most valuable remedies we possess for sick headache;



he thinks that it is most useful in preventing the attacks, not in arresting them when they have once begun. From a quarter to half of a grain of the extract or ten minims of the tincture may be given three times a day. It may be combined with iron or aloes. Caffeine is another remedy which appears to act by stimulating the vaso-motor nerves. It may be given in the form of strong infusion of the ground bean; but it is much more effectual to give the alkaloid itself. A citrate of caffeine, which is merely a mechanical mixture of caffeine and citric acid, is a useful mode of administration, or the alkaloid may be given subcutaneously. The citrate of caffeine may be given in doses of one grain every hour for some time before the expected paroxysm. Guarana has been recommended by Dr. Wilks, and I have occasionally found it useful, but as its active principle is identical with caffeine it is probable that it is not in any way superior to coffee or its alkaloid.

Nitrite of amyl produces a beneficial effect in the sympathico-tonic form of the disease. Berger was the first to employ nitrate of amyl inhalation in hemicrania, in a case of the spastic variety, and the pain vanished almost instantly, and did not return that day. Other observers have since found that this agent produces a temporary palliation, but that the pain returns in most instances after some time. Considerable care is requisite in inhaling the nitrite of amyl. It will be sufficient to inhale one or two drops on a handkerchief at first, and then the dose may be increased to three or five drops, and if necessary the inhalation may be repeated after a short time. Meyer recommends the inhalation of carbonic oxide gas, which also paralyses the vaso-motor nerves, but great caution is necessary in using so strong a poison. Croton chloral hydrate is a useful remedy, and although it does not always arrest the paroxysm of migraine, it is, so far as my experience goes, the most generally useful remedy which can be administered during the attack. I always give it in five grain doses every four hours till relief is obtained; but it may be administered in one large dose of fifteen grains.

Chloral hydrate, bromide of potassium, and chloride of ammonium may also be given during the attack, the latter agent being a particularly valuable remedy in many forms of head-

ache. Dr. Anstie thought the best means of arresting a sick headache was to give twenty grains of chloral, and make the patient plunge his feet into very hot water and mustard and breathe the steam. Morphia and other narcotics may be employed, but their effects in migraine are not so strikingly beneficial as in neuralgia.

The constant galvanic current is one of the most powerful remedies which we possess in hemicrania. Holst was the first to carry out a rational and methodical method of applying it. According to this method, one electrode, which is made long and narrow and with a considerable surface, is placed at the inner edge of the sterno-cleido-mastoid muscle, over the cervical part of the sympathetic, the other being placed on the palm of the hand. The pole on the neck is made positive in hemicrania sympathico-tonica, from ten to fifteen elements being used, and the current is suddenly closed, and after a passage of two or three minutes it is gradually stopped. The pole on the neck is made negative in hemicrania angio-paralytica, and the current is not only suddenly closed, but it is made to produce powerful excitations by means of repeated closures and openings, or in some cases by reversals. This treatment usually brings a sense of comfort and relief in a very short time, and in some cases it appears to produce a lengthening of the intervals between the attacks. Other authors prefer to pass the constant current continuously through the head.

The induced current has been recommended by Frommhold and Fieber. The former prefers the primary induced current, and applies one of the poles high up the back of the neck in the median line; and the other upon the forehead, or over the superciliary arch. Fieber employs the "electric hand." The patient takes one conductor in his hand, and the operator holds the other in his left hand while he applies the palm of his right hand firmly upon the patient's forehead, which is previously wetted.

## 2.—GRAVES' DISEASE (MORBUS BASEDOWII, EXOPHTHALMIC GOITRE).

The three prominent symptoms of Graves' disease are palpitation, enlargement of the thyroid gland, and exophthalmos.



§ 319. *Etiology*.—Sex is one of the most powerful predisposing causes of the disease, inasmuch as it affects the female twice as often as the male sex.

With regard to age, the middle period of life, between puberty and the climacteric period, is affected with especial frequency. The disease is rare in childhood; yet it has been met with in a girl of two and a half years. Rosenberg observed it in one of seven years, Solbrig in a boy of eight, and Trousseau in one of fourteen years. It is rare beyond the climacteric period, yet Stokes observed the disease in a woman of sixty years of age.

Hereditary predisposition appears to exert a certain amount of influence in the production of the disease. In the case just mentioned, as having been related by Solbrig, in a boy of eight, the mother is said to have suffered from the disease. But if the influence of direct inheritance is doubtful, there can be no doubt of the influence exerted by a neurotic predisposition. The disease is frequently associated with hysteria, epilepsy, and mental diseases.

Chlorosis and anæmia with their associated menstrual disturbances have been supposed to act as powerful factors in inducing the disease; but it is much more probable that the anæmia of Graves' disease is the result, and not the cause of the affection.

Various kinds of injuries have been known to have produced the disease. The most usual of these are mental excitement, violent fright, and injuries to the head. Climate may possibly exert some degree of influence in the production of the affection. Lebert states that it is more common in North Germany than in Switzerland and France, and it appears to be more frequent in England than on the continent.

§ 320. *Symptoms*.—The disease usually develops slowly and gradually, but when it is caused by emotional disturbances or injuries, it may begin suddenly, all the symptoms appearing in a few days. Peter describes the case of a woman, observed by Trousseau, in whom the disease was developed in the course of a single night as a result of profound grief for the death of her father. Her nose bled profusely during the whole of the night referred to.

In a few cases of sudden origin the disease runs an *acute*

course, and recovery may take place in a short time. In the boy observed by Solbrig recovery was complete in ten days.

Graves' disease may sometimes be preceded by various nervous symptoms, like those of hysteria or epilepsy. Palpitation is usually the first symptom of the disease, and occurs at first only at intervals, but after a time becomes habitual. The pulse is always much accelerated. In the lighter cases it may average from 90 to 120 beats per minute, but it may reach 200 beats, and at times it may be so rapid that it cannot be counted. The ordinary remedies for lowering the pulse often produce no effect. The palpitations and accelerated pulse are occasionally absent. Physical examination of the heart occasionally reveals a systolic murmur, which is probably often indicative of anæmia.

Other phenomena which are usually present are epigastric and occasionally hepatic pulsation, increased beating and visible pulsation in the carotids and their larger branches, especially the thyroid arteries, while a soft blowing murmur may be heard in them on auscultation even before the swelling of the thyroid gland is developed. The retinal arteries may be seen to pulsate on ophthalmoscopic examination.

Lebert regards the hepatic pulsation as arterial, and it is most frequently felt over the right lobe of the liver; hence it is not likely to be mistaken for the epigastric pulsation.

When the symptoms of disordered circulation have lasted for some weeks or months, the swelling of the thyroid gland, which forms the second great feature of the disease, becomes developed. It usually appears in the form of a soft elastic swelling, uniformly distributed over the whole thyroid gland. Occasionally one lateral lobe only is affected; and at other times, although both lobes are involved, one is more swelled than the other. The swelling occasionally appears before the palpitation, and still more rarely it is absent altogether. The surface of the tumour is generally covered by dilated veins, and a distinct thrill is communicated to the palm of the hand when it is laid upon it; and auscultation reveals loud blowing sounds, generally increased during the cardiac systole. These symptoms may diminish or disappear, while the tumour assumes a somewhat firmer consistence. The swelling of the



thyroid gland is seldom large, and the size of the tumour is subject to frequent changes, the swelling being increased by emotional disturbances and during pregnancy. In some cases each attack of palpitation is accompanied by enlargement of the thyroid gland, which disappears when the attack is ended; while in other cases the tumour may be observed to increase and decrease according to the force of the beat of the heart.

*Exophthalmos* makes its appearance soon after the swelling of the thyroid gland, and occasionally before it, and still more rarely it may precede both the palpitations and the swelling. The *exophthalmos* is almost always bilateral, but sometimes appears earlier in one eye than in the other, and often it is not equally developed on both sides. In a case observed by Dr. Yeo the enlargement of the thyroid and the *exophthalmos* presented a decidedly unilateral character; the goitre being on the right, and the *exophthalmos* on the left side. In some few cases the *exophthalmos* is absent, while in other cases it forms the only symptom of the disease.

The degree of *exophthalmos* varies greatly. At times there is only a slight prominence of the eyeball; while at other times it is so excessive that no part of the globe is covered by the eyelids, and even luxation of the globe may take place. The prominent eyeball has an unusual lustre, caused partly by the prominence of the globe, and partly by the fact of a rim of the sclerotic coat being visible round the whole of the cornea. If the deformity be of long standing, there is either partial or complete loss of mobility of the eyeball, which gives a peculiar stiffness to the expression of the patient. The *exophthalmos* may sometimes be diminished by slight pressure on the eyeball; at other times it increases or disappears proportionally to the force of the pulsations of the heart.

Von Graefe has pointed out that in the act of looking up or down the upper lid loses its power of moving in harmony with the eyeball, a phenomenon which must be regarded as a very valuable diagnostic sign of the disease. This symptom had, indeed, been previously described in graphic language by Charlotte Brontë in *Shirley*:—"Certainly Miss Mann," she said, "had a formidable eye for one of the softer sex; it was prominent, and showed a great deal of the white, and looked

as steadily, as *unwinkingly*, at you as if it were a steel ball soldered in her head." Dr. Gowers has shown that the movements of the eyelids are partly independent of direct muscular action. The cornea being more convex than the rest of the globe, a slight sulcus is formed where it joins the sclerotic coat. The rims of the eyelids, especially that of the upper one, are closely adapted to this sulcus, as they cross along the superior and inferior margins of the cornea, and when the eyes are directed downwards the edges of the superior lids maintain their position in the sulci, and the lids are therefore dragged down over the eyeballs, and, conversely, they are thrust upwards when the visual axes are directed to objects above the horizontal line. When the eyeballs become prominent the normal relations between the corneal sulci and the margins of the eyelids are lost, and it may be supposed that what may be called the mechanical movements of the eyelids cease, although the purely muscular movements may still be retained. But this explanation is insufficient, because the movements of the lids, as has been pointed out by Von Graefe, are unaffected in the exophthalmos which results from tumours of the orbit and other causes.

Disturbances in the nutrition of the globe occur in consequence of the exophthalmos and the defective power to depress the upper lid. In ordinary cases the conjunctiva alone is affected; it becomes dry, the veins become distended, and attacks of conjunctivitis supervene. In severe cases the nutrition of the cornea suffers, the symptoms being somewhat similar to those of neuro-paralytic ophthalmia.

The power of *accommodation* is usually unaffected in this disease, although it is sometimes weakened, owing to deficient mobility of the globe. Ophthalmoscopic examination generally shows a dilatation and increased tortuosity of the retinal veins, along with visible pulsation in the arteries. The pupil is generally unaltered.

Slight elevation of the temperature of the body can generally be detected by careful measurement. Paul, Teissier, Chendle, and Eulenburg have found an elevation varying from  $0.5^{\circ}$  to  $1^{\circ}\text{C}$ ; while Charcot and Dumont, on the other hand, report cases in which the temperature was entirely normal. Patients generally suffer from a sense of heat which is out of proportion to the



actual elevation of temperature, and it is sometimes so distressing as to lead the patients to throw off their clothes. This sense of heat is often accompanied by an increased secretion of sweat. Reynaud has directed attention to the connection between exophthalmic goitre and leucoderma. In five cases collected by him patches of leucoderma appeared on various parts of the body.

Graves' disease is sometimes complicated, as already noticed, with other severe nervous affections, such as hysteria, epilepsy, and insanity; but, in the absence of these grave disorders, patients manifest a changeable and emotional disposition, being subject to alternations of excitement and depression.

Patients often complain of severe headache, sometimes limited to one side, a feeling of dizziness, weakness of thought and memory, along with inability to work and sleeplessness. Usually there is complete anorexia, occasional vomiting and consequent emaciation, but in some few cases the appetite is excessive.

With the exception of the few cases which run an acute course, the duration of the disease is always very protracted. Sometimes complete recovery occurs either spontaneously or as the result of treatment. In some few cases pregnancy appears to have had a favourable influence on the course of the disease. Usually, however, the symptoms become gradually worse. The increased action of the heart leads to consecutive enlargement of the organ, with its usual deleterious effects. At other times death occurs from exhausting marasmus or some intercurrent disease, such as œdema of the lungs, apoplectic attacks, tuberculosis of the lungs, or valvular disease of the heart. In two cases death was caused by progressive gangrene of the lower extremities supervening without any apparent cause.

*Morbid Anatomy.*—The parenchyma of the thyroid gland is either normal, in a state of simple hyperplasia, or may contain cysts. The arteries have been found considerably developed, and the veins greatly dilated. A considerable development of fat is generally found in the orbit behind the globe, the ophthalmic artery is often atheromatous, while advanced fatty degeneration of the ocular muscles has been met with, probably caused by disuse and stretching.

Morbid changes have been described by various authors in the

cervical sympathetic and its ganglia, while other competent observers have failed to detect any appreciable alteration.

The principal alterations found were excess of connective tissue and a diminution of the nerve elements (Lancereaux), enlargement of the middle and lower cervical ganglia and disease of the cord of the sympathetic (Beveridge), obliteration of the lower cervical ganglia of both sides (Cruise and M'Donnell), atrophy of the sympathetic and its ganglia (Traube and Recklinghausen, and Biermer), and ensheathing of both cervical sympathetics in dense connective tissue (Geigel).

On the other hand, Wilks, Paul, Fournier, Ollivier assisted by Ranvier, and Rabejac assisted by Bouvier, found nothing abnormal in the cervical ganglia or in the cervical, thoracic, or abdominal cord of the sympathetic. In two cases which I examined with the greatest care, using a large number of stained preparations, nothing abnormal could be detected in the cervical sympathetic and its ganglia.

The results obtained so far by post-mortem examination are therefore somewhat doubtful; all that can with safety be concluded from the recorded examinations is that morbid anatomy favours the view that the anatomical substratum of the disease has its seat in the cervical sympathetic and its ganglia, supposing that this opinion can be supported by other evidence of an independent character. It is worthy of remark that in the few cases where positive changes were found in the sympathetic, the inferior cervical ganglion is the one which has been described as being chiefly or exclusively affected.

\* § 321. *Morbid Physiology.*—The theory of the disease has undergone considerable changes in the course of time. The disease was regarded by Basedow and other early observers as being the result of a morbid crasis, like that of chlorosis, and Stokes thought that the affection was due to disease of the heart. Laycock and various other authors regarded the affection as a neurosis, while Von Graefe, Aran, Trousseau, and others attributed the symptoms to disease of the cervical sympathetic, or of the cervical portion of the spinal cord and the medulla oblongata.

The origin of the swelling of the thyroid gland may be regarded as due to a dilatation of the arteries and veins of the



thyroid gland. Boddaert produced a swelling of the thyroid gland in rabbits and guinea-pigs by tying the internal and external jugular and the inferior thyroid veins. That the blood-vessels of the thyroid gland are dilated in this disease has been proved by post-mortem examination, and this is also shown to be the case during life by the fact that the small branches of the carotid and the thyroid arteries are tortuous, prominent, and may be seen and felt to pulsate strongly, that a soft blowing sound is audible over them, and that the tumour becomes alternately diminished or increased in size according to the force of the pulsation of the heart. The soft consistence of the tumour and its rapid growth point to the same conclusion.

Paralysis of the vaso-motor nerves which run in the cervical sympathetic is supposed to be the cause of the dilatation of the blood-vessels. It must be acknowledged, however, that there is no experimental evidence as yet that section of the sympathetics can produce the swelling of the thyroid gland. Benedikt thinks that the dilatation of the vessels is due to irritation of the dilator nerves, which run in the sympathetics, and not to paralysis of the vaso-motor, but the evidence for this theory is as yet insufficient.

The exophthalmos is probably due to various co-operating factors. It is in a great measure dependent upon increased development of fat in the cellular tissue of the orbit, and upon venous hyperæmia. Various reasons might be adduced to show that venous hyperæmia of the vessels of the globe and orbit exists during life, but it will suffice to mention that Boddaert has recently produced a considerable exophthalmos in rabbits and guinea-pigs, which lasted some days, by tying the two internal and external jugular veins, and dividing both cervical sympathetics.

That there is an increase in the fatty tissue behind the eyeball has been proved by a series of autopsies, and in one of two cases which I examined this was certainly the case. It is possible that contraction of the smooth muscles of the orbit discovered by H. Mueller, and which are innervated by the sympathetic, may assist in causing the exophthalmos. Claude Bernard has shown that section of the cervical sympathetic, or of the anterior roots of the two upper dorsal nerves, produced retraction of the

eyeball, while galvanic stimulation of the peripheral ends of the divided nerves caused dilatation of the palpebral fissure and exophthalmos. The exophthalmos in this experiment is probably caused mainly by contraction of Mueller's muscle. The action of the latter is rendered more effective in this disease inasmuch as the straight muscles are frequently in a state of fatty degeneration.

Irritation of the accelerator nerves might be supposed to be the cause of the increased cardiac action, but then it is necessary to assume a paralysis of the sympathetic in order to account for the swelling of the thyroid gland and the exophthalmos, and it appears a somewhat strained interpretation to assume that one set of fibres is irritated while another is paralysed by the same lesion. But affections of peripheral nerves sometimes give rise to symptoms of paralysis and of irritation in the same nerve. In neuritis, for instance, there may be symptoms of irritation in the motor fibres along with diminished sensibility, and, conversely, hyperæsthesia along with motor paralysis. It is not, therefore, impossible that some such conditions may exist in the fibres of the sympathetic, although few will accept this explanation as satisfactory. Benedikt endeavoured to overcome the difficulty by supposing that the thyroid swelling was caused by active irritation of dilator nerves which run in the sympathetic, but this supposition encounters many difficulties. It is, indeed, difficult to imagine that a nerve fibre would remain in a permanent state of irritation for many years without paralysis supervening. Friedreich, on the other hand, interprets the palpitations as a symptom of paralysis of vaso-motor nerves. Paralysis of the vaso-motor nerves of the sympathetic is followed, according to this view, by dilatation of the coronary arteries, increased flow of blood to the muscular walls of the heart, and increased excitement of its ganglia. This supposition surmounts the difficulty of supposing that a permanent lesion gives rise to continuous irritation without being followed by subsequent paralysis, but considerable difficulties stand in the way of both theories.

Some authors have endeavoured to explain the phenomena of the disease on the supposition that the cervical part of the spinal cord and medulla oblongata are the seat of morbid



changes. Geigel assumes that the lesions in the affection are to be found in the cilio-spinal region of the cord, and thinks that one of the centres—the oculo-pupillary—is in a state of irritation, and the other—the vaso-motor—is in a state of paralysis. Benedikt, on the other hand, places the lesion in the medulla oblongata. These theories are mentioned here for the purpose of directing the attention of future workers to the subject, so that further observations may be made which will either refute or confirm them.

*Diagnosis and Prognosis.*—At the beginning of the disease the diagnosis presents considerable difficulties. Provided the exophthalmos be bilateral and accompanied by general disturbance, the existence of the affection may be inferred even in the absence of one of the other two leading symptoms. Of the three leading symptoms one or two may be absent, or may disappear temporarily. The most valuable of the subordinate symptoms for diagnostic purposes are the feeling of heat, the elevation of temperature, and the want of agreement between the movements of the eyelid and globe. The cornea being completely surrounded by a visible ring of sclerotic is one of the most valuable indications of the slighter degrees of exophthalmos.

Cases of primary enlargement of the thyroid gland with consecutive irritation of the sympathetics may closely simulate Graves' disease, but in the former the pupillary and vascular symptoms are generally unilateral.

*The prognosis* is unfavourable, although some cases have disappeared spontaneously, or improved under medical treatment. The prognosis is more favourable when the general health of the patient is good, and when there is no organic disease of the heart.

§ 322. *Treatment.*—When the disease was regarded as being due to anæmia, quinine and iron were the remedies usually employed in its treatment; and although this theory is now abandoned, the treatment is by no means without value.

Remedies which depress the pulse like digitalis and veratrine have been prescribed in the disease, but they do not appear to possess any value. Iodine is said to diminish the swelling of

the thyroid gland, but to increase the palpitations. Belladonna has been employed with benefit.

Galvanisation of the cervical sympathetics has been used of late years in the treatment of the affection. Von Dusch treated a case for a considerable time in this way, with the result of reducing the rate of the pulse from 130 to 70 or 64 beats, and diminishing the exophthalmos. Eulenburg speaks favourably of this treatment. The method he adopted was to apply the negative pole to the cervical ganglia of each side alternately, or to both at once, by means of a divided electrode. He also attacked the swelled neck by *galvano-puncture*, but only doubtful results were obtained. Chovstek, Meyer, and Leube each obtained favourable results by galvanisation of the cervical sympathetics.

The habits of the patient should be carefully regulated; all fatigue, venereal excess, and mental excitement should be avoided. The diet should be mild and nutritious, chiefly composed of milk and vegetables, and the patient should avoid the use of alcoholic beverages, and even tea and coffee except in very moderate quantity. A large portion of the day should be spent in the open air, especially in the country, or in mountain health resorts of moderate elevation. Chalybeate mineral springs, such as those of Franzensbad, Pyrmont, Schwalbach, are of considerable value in the treatment of the disease. For the exophthalmos Von Graefe recommended painting with tincture of iodine, or friction with iodide of potassium ointment over the eyebrows and upper lids, compresses, local electrification, and in severe cases tarsorrhaphia as a protection against malignant affections of the cornea.

### 3.—UNILATERAL PROGRESSIVE ATROPHY OF THE FACE (HEMIATROPHIA FACIALIS PROGRESSIVA.)

Unilateral progressive atrophy of the face is characterised by a slow and gradual loss of substance in one side of the face, which usually begins in the external soft parts and passes successively to the deeper tissues.

*History.*—The disease was observed by Parry in 1825; but it was first carefully studied and described by Romberg and his scholars—Bergson, Schott, Heuter, and Axmann. Cases of the



disease were afterwards reported by Virchow, Samuel, Guttman, Moritz Meyer, and Moore. Although the disease was described by Lasègne, Ball, Aug, and Ollivier, yet Bitot observed the first case of the affection in France in 1862, and another in 1866; and his pupil, Lande, based upon these his inaugural dissertation in 1869, and his very able monograph published in 1870.

§ 323. *Etiology*.—The disease has hitherto been observed about twice as often in women as in men. In thirteen cases collected by Eulenburg the disease began before thirty years of age, the majority appearing between ten and fifteen years, but in a few instances the affection began as early as between two and three years of age. The affection appears to have a special predilection for the left side of the face.

In a few cases the outbreak of the disease has been preceded by scarlatina, measles, whooping cough, or a local herpetic eruption; and in other cases its origin has been attributed to exposure to cold. Toothache, and tearing pains in the head and the superior maxillary region of the affected side, have sometimes been complained of before the commencement of the atrophy. In a case published by Meyer, the patient suffered from epileptic fits, and some of these were limited to the portion of the face which afterwards became atrophied. In Brunner's case epileptic attacks supervened for the first time during pregnancy, and continued a year before the appearance of facial atrophy. The disease was preceded in Parry's case by left hemiplegia; and in the case observed by Heuter and Axmann the patient suffered, for many years before the atrophy began, from irregular spastic contractions of the masticatory muscles, along with some degree of hyperæsthesia of the left side of the face, that being the side which was afterwards the subject of atrophy. Anjel also observed a case in which spasms, hyperæsthesia, and paræsthesiæ in the left half of the face preceded the atrophy.

§ 324. *Symptoms*.—The first symptom to attract notice is a peculiar discolouration of circumscribed areas of the skin. A white spot appears on one side of the face; it is slightly depressed and spreads. Several of these spots appear sometimes, either simul-

taneously or in succession, and after a time they coalesce so as to form a spot of considerable size. The white colour may be transitory, and the affected area may assume a yellowish or brownish tint, such as is frequently observed in cicatrices after burns. These spots soon become the seat of a marked atrophy, the skin becomes thin and emaciated, the subcutaneous fat disappears so that the side of the face becomes deformed by pits of greater or lesser size and depth. The beard, eye-lashes, and even the hair of the head on the affected side usually undergo structural changes, and these may sometimes precede the formation of the cutaneous spots and depressions. The hair at times turns perfectly white, at other times it falls out, or its growth is more or less interfered with, while in a few cases streaks only of the hair of the head or of the lids and brow are discoloured. The secretion of the sebaceous follicles is arrested, but the functions of the sweat glands appear to be normally performed. The involuntary muscular fibres of the skin give a normal reaction to electrical stimulation. In advanced cases the affected skin feels irregular, atrophies, and may assume the form of a cicatrix, but it does not become adherent to the underlying structures. The cutaneous sensibility is not usually affected, but in one case it was diminished, while in other cases hyperæsthesia has been observed. Abnormal sensations have often been complained of in the atrophied portions of skin. Neuralgic attacks have been experienced on the affected side during the progress of the disease, but pain is not an invariable symptom. The muscles on the affected side are, as a rule, unaffected, and give normal reactions to electrical stimulation. In some cases the muscles were diminished in bulk, fibrillary twitchings were observed, and the face was somewhat drawn to the atrophied side.

In a case observed by Eulenburg and Guttmann, the masticatory muscles on the affected side were distinctly emaciated, and the movements of mastication on that side were weakened. In other cases distinct asymmetry in the contour of the upper lip has been observed, owing to atrophy of one-half of the orbicularis, while in other cases the half of the tongue on the affected side was diminished in bulk; the vault of the palate, the velum and uvula have also at times been involved in the



atrophy. Deglutition has not been interfered with in any recorded case; but in one case the pronunciation of the letter "r" was somewhat impeded.

The annexed figure, borrowed from Romberg, represents the case of an unmarried woman, aged 28 years, who had suffered from this affection. The left side of her face had gradually atrophied as the result of extensive suppuration on the left side of the neck, which burst through the tonsil. Every feature, including the brow, eye, nostril, lips, cheek, and

FIG. 99.



chin, as well as the left half of the tongue and left palatine arch, was smaller than those on the opposite side.

*The large arteries* of the face are, as a rule, unaltered in size, and the tone of the small arteries is retained or even increased. The atrophied parts are generally capable of blushing, and they also redden under local electrical excitation. The temperature is the same on both sides.

*The bones* of the face have been found distinctly diminished in volume. Both the upper and lower maxilla and malar bones have been implicated; the cartilages of the nose have also been observed diminished in bulk; and the teeth may undergo consecutive alterations. The fatty tissue behind the globe

often disappears, so that the eye on the affected side falls back into the orbit and appears more sunk and smaller, and the opening of the lids narrower than on the sound side. The disease is always slow and protracted, and its course is usually steadily progressive, although a brief pause appears in some cases. The general health is not interfered with.

§ 325. *Pathology.*—The majority of authors regard the disease as of nervous origin, although Lande and a few others believe it to be a *primary* atrophy of the fatty tissue and of the cells and fibrils of the connective tissue, leaving only the elastic tissue unaltered.

The nervous theory of the origin of the disease has assumed two chief forms, some pathologists attributing the affection to disease of the vaso-motor, and others to disease of the trophic fibres of the trigeminus. Bergson was the first to attribute the disease to a primary affection of the vaso-motor nerves. In the case described by him, the carotid artery on the affected side pulsated less strongly than that on the sound side, and he thought that the atrophy was due to the diminution of the calibre of the artery. But this theory is manifestly inadequate, since neither continual irritation nor paralysis of the vaso-motor nerves gives rise to symptoms at all comparable with those of progressive paralysis of the face.

Romberg classified this affection as a trophoneurosis, but the relation of the disease to the trophic system of nerves was afterwards more fully developed by Samuel. Atrophy of the muscles supplied by the motor portion of the fifth has been observed in some cases of the disease, a circumstance which strongly points to the influence of the fifth in the production of the affection. Muscular atrophy, however, is always a subordinate symptom in comparison with the nutritive disturbances of the integuments, the former being in many cases completely absent.

Experiments have shown that the vaso-motor and trophic fibres of the face run in the sympathetic before passing to the trigeminus, hence it is possible that this disease may at times be caused by lesion of the sympathetic. Seeligmueller described two cases in which the atrophy appeared to be due to injury



of the sympathetic. In the first case the cord of the sympathetic and the brachial plexus in a child were injured by fracture of the clavicle and neck of the scapula, and myosis was combined with decided atrophy of the side of the face. The second case was one of gunshot wound of the left sympathetic and a part of the brachial plexus, and in addition to the usual oculo-pupillary symptoms there was distinct emaciation and flattening of the left cheek. In a case observed by Brunner, the symptoms corresponded to the usual results of experimental irritation of the divided cervical sympathetic. The symptoms were dilatation and deficient reaction of the pupil, widening of the palpebral fissure, exophthalmos, deficiency of the lachrymal secretion and of the secretion of mucus and sweat, and lowering of the temperature of the whole left side of the face. The left superior ganglion of the sympathetic was tender on pressure. Brunner thinks that a continued irritation of the sympathetic produced a continued spasm of the blood-vessels, and that this led to a gradual atrophy of the left side of the face.

§ 326. *Diagnosis and Prognosis.*—The disease may be mistaken for congenital asymmetry of the two halves of the face, but in the latter condition the characteristic atrophy of the skin and subcutaneous tissues are wanting. In torticollis and scoliosis of the vertebral column, the side of the face corresponding to the convexity of the main curve is often smaller than the other side. Arrest of development of one side of the face may be caused by traumatic injury received during youth, but neither the colour of the face nor of the hair is altered. Hypertrophy of the opposite side of the face can only be mistaken for atrophy by carelessness of observation. Certain cutaneous affections, as vitiligo and tinea decalvans, may be mistaken for the early stage of the affection. In vitiligo there is the same white discolouration of the integument, the cicatricial appearance of the skin, greyness and falling out of the hair; but the loss of volume, which is the special characteristic of progressive unilateral atrophy, is absent. In tinea decalvans the disease first appears in regular circular spots, it is contagious, fungi can be detected, and the hairs fall out without previous loss of

colour, and the loss of hair is preceded by inflammatory symptoms and oedema of the skin.

Unilateral atrophy of the face is not dangerous to life, nor is the general health affected, but so far as recovery is concerned the prognosis is very unfavourable.

§ 327. *Treatment.*—Various remedies have been employed, but none of them have hitherto proved successful. Electrical treatment has been adopted in almost all the reported cases. The use of the constant current is said to have produced in some cases an improvement in the nutrition and colour of the skin, and under its use the power to blush returned. In a case observed by Eulenburg and Guttmann, the local application of the faradic and galvanic currents for several months and the galvanisation of the sympathetics did not lead to any permanent benefit, though local galvanisation produced a reddening of the affected side of the face, which continued for several hours after each application.



## CHAPTER III.

DISEASES OF THE THORACIC PORTION OF THE  
SYMPATHETIC.

## ANGINA PECTORIS.

ANGINA PECTORIS consists of paroxysms of pain in the præcordial region, usually radiating over the left side of the thorax and down the left arm, and associated with a peculiar sensation of constriction and intense anxiety. It must be remembered that every form of angina pectoris is not due to diseases of the sympathetic nerves; but, as the different varieties of the affection are allied clinically, it is deemed better to describe all of them in this place.

§ 328. *Etiology*.—Angina pectoris is sometimes a mere symptom of grave organic disease, such as aortic disease, fatty degeneration of the heart, and ossification of the coronary arteries, but such cases cannot be discussed here. The only cases which will be mentioned here are those in which the angina is a pure neurosis, unaccompanied by any perceptible organic change in the heart. The causes of the neurotic affection are singularly obscure. Hereditary predisposition can be traced in many cases of angina, and it is frequently found in members of families who manifest a tendency to other neurotic diseases, such as hysteria, insanity, and epilepsy. Attacks of angina may form a symptom of hysteria, precede or alternate with an attack of epilepsy, or constitute an intercurrent symptom of chronic mental disease.

The disease occurs more frequently in advanced age, but it is probable that the proportionate frequency with which it is supposed to occur in old age depends upon the angina symptomatic of organic cardiac disease being mistaken for true neurotic angina pectoris. The male sex is much more liable to be attacked than

the female, probably because the former is more exposed to the exciting causes than the latter.

The first of the exciting causes of the disease which deserves mention is exposure to cold. Excessive smoking of tobacco appears undoubtedly to induce attacks of the disease. Beau describes eight cases in which the attacks ceased when smoking was stopped, and returned on the practice being resumed, and similar cases are recorded by other authors. Other exciting causes are certain morbid conditions of the thoracic organs, which involve the nerves of the cardiac plexus, and morbid conditions of the abdominal regions, which probably act by reflex irritation.

§ 329. *Symptoms*.—Angina pectoris occurs in paroxysms, which are separated from one another by longer or shorter intervals. The attack begins suddenly. Pain is felt at the lower part of the sternum, and shoots over the left side of the chest and neck, or along the sternum and down the left arm or both arms. When both arms are implicated, the pain is always more pronounced on the left side of the chest and left arm than on the corresponding parts of the right side. The character of the pain is described as shooting, tearing, aching, sickening, or burning, and it is often said to be indescribable. A feeling of oppression or constriction is felt across the chest as if it were being forcibly compressed and could not be expanded. This feeling is accompanied by a sense of suffocation and inability to breathe, although the patient is only prevented from breathing by the pain, and not from any mechanical interference with the act of respiration. The pain is accompanied by a feeling of great anxiety and dread of impending death, the face becomes pale and covered with sweat, and the expression is one of great alarm and fear. The beats of the heart are usually feeble and intermittent; while at other times the action is violent and forcible, but this occurs more frequently towards the end of the attack. At the beginning of the attack the arteries are like cords, the pulse small and wiry, but in the course of the attack it becomes feeble, fluttering, and irregular, and in the remissions it becomes full and soft. Sphygmographic tracings show an increase of arterial tension at the beginning of the attack, and later a lessened tension.



During a paroxysm of angina the skin is generally pale, cold, dry, that of the hands and feet almost bloodless, the face pale and sunken, the patient feels chilled, and the teeth chatter; but towards the end of the attack the opposite condition obtains, the surface becomes warm and red, and the skin covered with abundant perspiration. The duration of the paroxysm is generally short, usually only a few minutes, but sometimes the attack is made up of a series of paroxysms, each being followed by a remission or a complete intermission of the distressing symptoms.

The course of the disease is always chronic. The attacks recur, as in epilepsy, at extremely variable intervals. At times the interval may extend over years, while in other cases the attacks recur for a time every day without any cause being recognisable. Great bodily and mental exertion may undoubtedly provoke an attack, and they may sometimes be excited or made worse by pressure on some points which are sensitive constantly or only during the attacks. These points are the spinous and transverse processes of the cervical and upper dorsal vertebræ, and the region of the inferior angle of the scapula. As time advances, the attacks usually grow worse and recur with greater frequency.

§ 330. *Morbid Anatomy.*—It would be out of place to refer here to the various organic diseases with which angina pectoris is associated; the only cases which come within the scope of this article being those in which changes have been discovered in the nervous apparatus of the heart. In a case of angina under the care of Skoda in 1841, the right phrenic nerve was found at the autopsy, which was made by Rokitanski, involved in a dark blue, hard knot, dotted with concretions of lime. The *cardiacus magnus*, rising from the cardiac plexus between the pulmonary artery and the descending aorta, was seen to enter into a black nodule of the size of a hazel nut. Upon the exterior aspect of the left bronchus the descending branches of the left vagus were observed similarly interfered with by a blackish lymphatic gland that lay beneath them. Dr. Haddon found the left phrenic nerve compressed by a bronchial gland of the size of a hazel nut, situated close to the root of the left lung. In

this case there was aneurismal dilatation of the aorta, so that it is very doubtful whether the compression of the phrenic had anything to do with the production of the angina. Lanceraux found microscopical changes in the cardiac plexus associated with an alteration of the aorta at the point where the cardiac plexus lies upon it; and in two other cases he found similar changes along with narrowing of the coronary arteries. But it must be acknowledged that in all these cases it is exceedingly doubtful how far the morbid change of the cardiac plexus had produced the symptoms, inasmuch as the symptoms might have been produced by the organic change of the aorta and of the coronary arteries. The results obtained from post-mortem examination with respect to diseases of the nerves, independently of disease of the structure of the heart, are, indeed, very scanty, and it would be precarious to draw definite conclusions from these.

§ 331. *Morbid Physiology.*—Laënnec regarded angina pectoris as a neuralgia of the nerves of the heart, and Trousseau as an epileptiform neuralgia; while Romberg and Friedreich attributed it to a hyperæsthesia of the cardiac plexus. Other authors, however, regarded the alteration in the action of the heart as the prime factor in the disease, and relegated the pain to a purely secondary position. Stokes, for instance, thought the symptoms were due to a temporary increase of weakness in an already weakened heart, and Donnes and John believed that the essential condition was a paresis or paralysis of the heart. Romberg thought the condition due to increased action of the heart, or to a hyperkinesis with hyperæsthesia; and Von Dusch regarded it as a hyperæsthesia combined with spasms of the heart. And opposite as these opinions may appear to be, all of them may be adumbrations of the truth. Eichwald, however, enunciated a more comprehensive formula for the condition which underlies angina, when he attributed the symptoms to over exertion of the heart, caused by some obstruction to the onward flow of blood. In order to give a more comprehensive expression to the hypothesis, it may be assumed that the essential condition which underlies angina is a disturbance between the balance which ought to be maintained between the propulsive powers of the



heart and the resistance to be overcome in such a way that the muscular walls of the heart are subjected to strain. This state may result from all conditions which weaken the muscular power of the heart, such as fatty degeneration; as well as from those conditions which cause obstruction to the onward flow of blood, such as aortic obstruction. But, as already pointed out, angina may often be quite independent of any organic disease of the heart, and in these cases we are driven to look to the nervous system for an explanation of the condition which causes the disruption between the propulsive powers of the heart and the resistance to the onward flow of blood. The idea that angina pectoris must be ranked amongst the vaso-motor neuroses occurred to several authors independently of each other. Dr. Lauder Burton in 1866 found from sphygmographic tracings that there was increased tension in the arteries during the attack, which he attributed to contraction of the small systemic vessels. This opinion led him to administer the nitrite of amyl by inhalation; the arterial pressure was soon lowered and the attack relieved. About the same time Landois subjected the symptoms of the affection to a systematic physiological analysis, and showed that it might be divided into several distinct varieties.

The following are the varieties of angina pectoris, which are at least theoretically possible:—

1. *Automatic Excito-motor Ganglia of the Heart.*—Certain poisons introduced into the ventricular cavity, or solutions into which the entire heart is plunged, are capable of immediately arresting the action of the heart, which has been removed from the chest and still pulsates. According to the experiments of Landois, weak solutions of these agents injected into the endocardium of the frog stimulate the ganglia, causing an increase in the rate of speed of the heart; while stronger solutions of the same agents paralyse the ganglia and arrest the action of the heart. Similar states of the ganglia may be induced by pathological conditions; and when these morbid conditions are such as to produce irritation of the ganglia, the rapidity of the heart's movements are increased, while if the influence exerted on the ganglia be a paralysing one the rapidity of the heart's movements is diminished; and there can be little doubt that angina is associated with the latter condition.

2. *Vagus angina pectoris* may assume two forms. (a) The first is produced by causes acting directly on the vagus. Irritation of the vagus is indicated by a full, hard, retarded pulse, increased force of the pulsations

of the heart, disturbance of phonation and deglutition, and sometimes temporary arrest of the action of the heart. Paralysis of the vagus is indicated by increased rapidity of the pulse. (b) The second form of vagus angina pectoris is induced by reflex causes, and it is especially frequent in diseases of the abdominal organs. Landois has called this form *angina pectoris reflectoria*. If the abdomen of a frog be laid bare and the intestine be struck sharply, as with the handle of a scalpel, the heart will stand still in diastole with all the phenomena of vagus inhibition, and the peripheral irritation which results from disease may produce an inhibitory action upon the heart.

3. *Excito-motor Sympathetic Angina Pectoris*.—This form is caused by an affection of the accelerator nerves of the heart, which run with the sympathetic, and the symptoms are the same as in the first form of the affection. The most probable seat of the lesion is the cardiac plexus.

4. *Angina Pectoris Vaso-motoria*.—Irritation of the vaso-motor nerves, which run for the most part in the sympathetic, causes contraction of the arterioles, and consequent increased resistance to the ventricular systole. Marey has pointed out that the rapidity of the beats of the heart is in inverse ratio to the arterial pressure; hence during irritation of the vaso-motor nerves the pulse will be slow and hard, and there will be paleness and coldness of the skin. During paralysis of the vaso-motor nerves, the pulse will be full and quick, and there will be warmth and redness of the surface; but it is very doubtful whether this condition ever induces the symptoms of angina, although it very generally forms a secondary stage of the affection after a primary stage of irritation.

The pain which forms such a prominent symptom of angina pectoris is due to neuralgia of the cardiac plexus; but like other forms of neuralgia it may be induced by various causes. Eichwald attributes the pain to the over-exertion of the heart to overcome an obstruction, just as over-action of other muscles, both voluntary and involuntary, is painful, such as cramp of the calf and contraction of the uterus. It is certain, however, that the pain may be produced by other causes, such as mechanical pressure, and other injuries of the cardiac plexus, and anemia of the cardiac nerves and ganglia due to ossification of the coronary arteries and disease of the aortic valves.

The pains which radiate from the præcordial region to other parts of the body, especially the thorax and arm, are rendered intelligible by the anatomical connections which subsist between the cardiac plexus and cervical nerves, through the medium of the cervical ganglia.

§ 332. *Diagnosis and Prognosis*.—The diagnosis resolves itself into the question whether there is organic disease of the heart, or whether the symptoms are due purely to a disturbance of the nervous system. This question must be determined by a careful physical examination, giving due weight to concomitant



circumstances, such as the age and general condition of the patient.

The prognosis depends to a considerable extent upon the gravity of the accompanying organic changes, but it is always grave in true angina pectoris.

§ 333. *Treatment.*—In cases where physical examination shows that the angina is symptomatic of structural disease of the valves of the heart or great vessels or on arterio-capillary fibrosis, the treatment resolves itself into that which is appropriate for the graver affection. When the symptoms depend upon abdominal disease or excessive tobacco-smoking, an endeavour must be made to remove the cause of the disease.

If the affection be of nervous origin, the treatment must be directed to remove the symptoms. The remedies most usually employed are diffusible stimulants and so-called antispasmodics, such as alcohol in its various forms, the various ethers, valerian, musk, castor, camphor, succinate of ammonia. Ether or chloroform inhalation may be cautiously employed, but without producing full narcosis. Narcotics are also useful, especially the subcutaneous injection of morphia. Atropine and conia have been recommended, but both appear to be inferior to morphia.

In the cases where vascular spasm is present, the inhalation of nitrite of amyl sometimes gives immediate relief. Such a powerful remedy requires, of course, to be used cautiously. In cases characterised by violent and accelerated action of the heart due to abnormal irritation of the automatic ganglia, or the excito-motor fibres of the sympathetic, such agents as atropine, conia, aconite, and veratria may be found useful.

In order to prevent a recurrence of the attacks, the action of the heart must be strengthened. Various remedies have been employed for this purpose, but probably iron, arsenic, and digitalis are the remedies which are most to be depended upon. Sulphate of zinc, cyanide of zinc, nitrate of silver, bromide of potassium, and bromide of calcium are other remedies which have been recommended by various authors, and which may occasionally be useful, although less applicable than the three remedies just mentioned. Quinine, phosphoric acid, inhalation of oxygen, and hydrocyanic acid have also proved serviceable

in certain cases. Counter-irritation of the cardiac region was at one time a favourite remedy, and it is quite possible that it is rather too much neglected in the present day. A great deal of benefit is found in some cases from covering the præcordial region with a belladonna plaster.

The induced current in the form of the faradic brush has been employed by Duchenne, who thought that he produced a permanent cure in two cases—one being a case of the uncomplicated nervous form, and the other hysterical. Cutaneous irritation by means of faradisation acts in a reflex manner on the nerves of the heart and the vaso-motor nervous system. Weak interrupted currents strengthen the contractions of the heart, accelerate the circulation, and contract the vessels by reflex excitation of the excito-motor and vaso-motor nervous apparatus; but strong faradic currents weaken the contractions of the heart, retard the circulation, and dilate the vessels by reflex excitation of the regulator nerves of the heart and paralysis of the vaso-motor centres. Severe cutaneous irritation, therefore, is only justified when the action of the heart is powerful, rapid, and violent, with contracted arteries and small tense pulse. The same considerations must direct our employment of galvanisation; the strong currents must be applied so as to influence the regulator nerves of the heart only when the action of the heart is quick and violent; while weak currents must be used to the cervical sympathetic and cervical vagus when the action is slow and feeble. When it is desired to act on the inhibitory nerves strong stable currents may be used; the positive pole should be placed on the sternum, while the negative is placed on the lower cervical vertebræ. In a case of supposed rheumatic origin, Von Huebner obtained a complete cure by placing the positive pole upon the fossa supersternalis, and the negative upon the cervical ganglia of the sympathetic on each side in succession. He placed the positive pole over the lowest cervical ganglion and the negative upon the sensitive spots at the angles of both shoulder-blades. At first weak currents were used, and the attacks ceased after the first sitting, and did not return.



## CHAPTER IV.

DISEASES OF THE ABDOMINAL PORTION OF THE  
SYMPATHETIC.

## (I.)—NEUROSES OF THE CÆLIAC PLEXUS.

*Neuralgia Mesenterica (Colic, Enteralgia, Colica Saturnina).*

§ 334. Enteralgia, or intestinal colic, consists of paroxysms of pain in the abdomen, having their seat of maximum intensity about the umbilical region.

*Etiology.*—Any source of irritation of the mucous membrane of the alimentary canal may give rise to colic, such as undigested or irritating food, cold drinks or ices, retained fæces, and the presence of gall stones or worms. The source of the irritation may be in the mucous membrane itself; hence colic is often a prominent symptom of organic diseases and obstruction of the intestines. The origin of the irritation may be remote and indirect; hence intestinal colic may likewise be a symptom of ovarian and uterine affections, or result from the passage of hepatic or renal calculi. Exposure to cold is a frequent cause of colic, and is probably produced in a reflex manner. Diseases in other parts of the nervous system may cause colic, and it is especially frequent as a symptom of hysteria. Intestinal colic is sometimes caused by certain morbid poisons in the blood, as gout and rheumatism; but chronic lead poisoning produces the severest and most obstinate form of colic, although the affection may be a symptom of chronic copper poisoning, and may probably be caused by other mineral poisons.

*Symptoms.*—Severe pains are felt in the abdomen, which usually occur quite suddenly, without any premonitory symptom. The pains are described as being of a twisting, pinching, or griping character. They begin and are most severe about the umbilical region, but may spread over the entire abdomen,

and are liable to change their position. The pains occur in paroxysms, which are followed by periods of remission or complete intermission. Pressure of the abdomen almost always affords relief, and the patient usually bends forwards and presses the abdomen with both hands, or lies on the abdomen. A diffused tenderness of the abdominal walls, on deep pressure, especially if suddenly applied, is not unfrequently experienced.

The bowels are usually constipated, and distended with flatus, so that a very tympanitic note is afforded on percussion, and the rolling about of flatus can often be felt when the hand is placed on the abdomen, while the abdominal muscles are felt contracted, hard, and knotty. Colic may occasionally be associated with diarrhoea. In lead colic the abdomen is retracted, there is obstinate constipation, nausea, vomiting, eructations, and hiccup; while the blue line on the gums will indicate the presence of the poison in the system.

Disorders of the circulation are usually associated with every form of colic. During the attack the face and extremities are pale and cold, and the action of the heart becomes slow and feeble, and if the attack be prolonged there may be symptoms of more or less complete collapse. The action of the heart is unusually weak in lead colic, and the pulse, although occasionally quickened, is as a rule rendered slow. Out of 1,217 cases collected by Tanquerel, the frequency of the pulse in 678 was only from 30 to 60 in the minute; and Eulenburg observed cases in which the pulse beat only 28 times in the minute. Cases have been observed, however, in which there has been increased frequency of the pulse. Romberg has drawn attention to the fact that patients suffering from intestinal and other forms of colic experience a subjective sensation of faintness and weakness, and a sense of impending death more or less similar to that which is experienced during attacks of angina.

§ 335. *Pathology*.—It is wholly unnecessary to enter here into a detailed account of the various opinions held with regard to the nature of colic, nor need we enter on the question whether it is to be regarded as a hyperæsthesia, or a neuralgia of the mesenteric plexus and its ganglia. Ludwig has found that the splanchnic nerves are highly sensitive to nervous impressions,



and, consequently, it is very probable that the painful impressions reach the sensorium through these nerves. Eulenburg and Guttmann suggest that the sensory fibres which surround the abdominal arteries may also be involved in the enteralgic attack. The disorders of the circulation are to be explained by supposing that the irritation of the intestines is conveyed by afferent fibres to the centre of the vagus in the medulla oblongata, and thence reflected outwards to the heart, inhibiting its action, as occurs in Goltz's percussion experiments with the intestines of the frog. It must, however, be remembered that Marshall Hall arrested the action of the heart of an eel by crushing the stomach after the brain and spinal cord had been entirely removed; so that the irritation must have been conveyed from the latter to the former without the intervention of the medulla oblongata.

§ 336. *Treatment*.—The first aim of treatment must be to remove the cause of the affection. As constipation is usually present, a free aperient must be administered, and probably the best for the purpose is a full dose of castor oil. If the pain be severe, twenty to thirty drops of tincture of opium may be given along with the oil. Other forms of aperient may do equally well, or the bowels may be evacuated by means of enemata of warm water. Carminative drinks, or some spirit and hot water, are useful during the attack, and if hysteria is associated with the attack a draught containing tincture of valerian or assafætida is indicated. For the relief of pain the greatest reliance must be placed, as usual, upon opium—either a full dose of the tincture or a subcutaneous injection of morphia. Fomentations over the abdomen or dry heat with friction are useful adjuncts in treatment, and if any signs of collapse supervene diffusible stimulants must be given more or less freely, according to circumstances. In lead colic the poison must be eliminated by means of vapour baths, diaphoretics like jaborandi, and above all by the administration of iodide of potassium, which renders soluble the lead which has been deposited in the tissues. The faradic and galvanic currents may be useful in the treatment of the affection, although their efficacy has not as yet been indubitably proved.

§ 337. *Spasm and Paralysis in the Region of the Celiac Plexus.*

The movements of the larger portion of the intestinal tract from the stomach to the transverse colon are regulated through the splanchnic nerves; and consequently the motor disturbances to which these organs are liable are in great part caused by disorder of the sympathetic system. The vomiting, which accompanies hysteria, neurasthenia, tabes dorsalis, and other nervous diseases, is in all probability due to implication either directly or indirectly of the sympathetic system. Reflex vomiting accompanies the passage of a gall stone or renal calculus, as well as other diseases attended by severe pain.

Irritation or paralysis of the regulatory fibres of the splanchnic nerves may give rise to obstinate constipation, or to an increased peristaltic action of the bowels. The obstinate constipation of lead colic may be taken as an example of the former, and the increased peristaltic action caused by various purgative medicines of the latter.

(II.)—NEUROSES OF THE GASTRIC PLEXUS.

Some of the sensory neuroses, as Pyrosis, Bulimia, and Polyphagia, which occur in the territory of distribution of the gastric plexus, have been already described, and it only remains to mention briefly the neuralgic attacks which occur in this region.

§ 338. *Neuralgia Gastrica (Cardialgia, Gastralgia, Gastrodynia Neuralgica, Neuralgia Cœliaca.)*

Gastrodynia is characterised by paroxysmal attacks of pain in the epigastric region, which may radiate upwards to the back between the shoulders, or towards the middle of the sternum. The attack usually comes on suddenly, without any premonitory symptom, and the pain, which is very severe, generally intermits after a few minutes, but soon recurs with greater intensity, and after repeated intermissions finally disappears. Tenderness of the epigastrium on pressure is not usually present, the pain being, on the contrary, often relieved by firm and continuous pressure. Pressure on the cartilages of the false ribs on the left side or on the corresponding intercostal space



may cause pain, and the spinous processes of some of the vertebræ may be tender on pressure. In severe cases a feeling of oppression and faintness accompanies the attack, due to an effect exerted on the circulation, and the affection is therefore closely allied to *angina pectoris reflectoria*.

During the attack the upper portions of the recti muscles are strongly contracted, and the abdominal walls consequently rendered tense and unyielding. The epigastric region is usually retracted, seldom swelled. The pulse is generally slow and feeble, the arterial tension low, the extremities cold and pale, and towards the end of a severe attack chattering of the teeth, faintness, and vomiting have been observed.

The duration of the attack varies from a few minutes to a few hours, or even to a few days, and during the interval the patient is free from pain. The recurrence of the attacks is very irregular; at times they occur daily, while at other times years may intervene between two attacks.

The etiology of the disease is obscure. The affection is more common in youth than in old age and in women than in men. It is often associated with *anæmia* and *chlorosis*, and is not an unfrequent manifestation of *hysteria*. Hereditary predisposition to the affection can occasionally be traced, and a depressed nutritive condition predisposes to *gastralgia* as to other forms of *neuralgia*. The stomach is free from structural change, and the digestion may in other respects be carried on satisfactorily. *Gastralgic* attacks are not unfrequent in certain diseases of the spinal cord, especially in *tabes dorsalis*.

*Diagnosis.* — The pain of gastric *neuralgia* occurs in paroxysms with perfectly free intervals, it occurs independently of the kind of food taken into the stomach, and it is relieved by pressure on the epigastrium, and the nutrition of the patient is rarely affected. In chronic structural diseases of the stomach, on the other hand, the pain is increased by the action of anything which irritates the mucous membrane of the stomach; it occurs soon after partaking of food, more especially if the food or drink contain anything pungent or irritating, it is increased by deep pressure over the epigastrium, and the patient loses flesh and becomes progressively emaciated, and assumes a *cachectic* appearance.

*Prognosis.*—The prognosis of gastric neuralgia is generally favourable, except when it depends upon hysteria, or a central lesion like locomotor ataxy.

*Treatment.*—The causes must first be removed. When it occurs in young, anæmic, and chlorotic females, the usual treatment of these affections must be adopted. If a malarial origin be suspected, quinine is likely to prove an effectual remedy. In many cases a cure is effected by the removal of other local diseases, as metritis, or ulceration of the os uteri. In the great majority of cases, the treatment must be palliative, and directed to allay the local pain. The most usual remedies for this purpose are bismuth, nitrate and oxide of silver, opium, or the subcutaneous injection of morphia. I have several times found the constant current useful, the positive pole being placed on the sternum, and a large negative pole on the epigastrium.

#### (III.)—NEURALGIA HEPATICA (HEPATALGIA).

§ 339. Hepatic colic is usually the result of the passage of biliary calculi through the cystic and common ducts. Paroxysms of similar pain, however, occur at times in neurotic subjects, in the entire absence of jaundice and the other symptoms which indicate the passage of gall stones, and when no other source of irritation can be discovered; hence these are regarded as of purely neuralgic origin.

*Symptoms.*—The symptoms of hepatic colic are more or less severe, pain coming on suddenly and lasting with irregular intermissions and exacerbations a few hours or a few days. The pain is often very severe, and of an aching, cutting, or tearing character, and is usually attended with a sense of constriction or cramp. It is generally referred to the pit of the stomach, or to the umbilicus, whence it radiates to the back between the shoulders, but never downwards (Murchison). The patient suffers during a severe attack from faintness, nausea, and vomiting, the action of the heart is weakened and the surface of the body is cold, and in severe cases there are symptoms of collapse.

*Treatment.*—The treatment of hepatic colic must be conducted on the same principles as for enteralgia, by fomentations and narcotics, and the removal of the exciting cause.



§ 340. *Addison's Disease (Bronzed Skin).*

The peculiar cachexia and pigmentation of the skin, which constitute the chief clinical features of Addison's disease, have almost always been found associated with disease of the supra-renal capsules. The semilunar ganglia and solar plexus are, however, frequently implicated in the disease, and some pathologists regard the affection of the sympathetic as the primary and fundamental anatomical change, and that upon which the symptoms depend. A large number of post mortem examinations might be cited in favour of this opinion; but so long as the nervous origin of the disease is uncertain, it will be better to refer the reader to other sources of information with regard to this affection.

§ 341. *Diabetes Mellitus.*

The vaso-motor nerves of the liver take their origin on the floor of the fourth ventricle, and pass through the cervical and upper dorsal regions of the spinal cord, and the *rami communicantes* opposite the fourth or fifth dorsal vertebra, to join the sympathetic, and ultimately enter the organ as the hepatic plexus. Injury to these fibres, either at their origin in the floor of the fourth ventricle, or in any part of their course in the spinal cord, or in the sympathetic itself, produces a paralytic dilatation of the vessels of the liver, that is followed by an increased formation of sugar in the organ, which is eventually eliminated by the kidneys. There can be no doubt that many forms of diabetes mellitus are similarly caused. Many cases of diabetes are caused by injury, or disease of the brain, involving the floor of the fourth ventricle, or the parts near it. Diabetes has also been found to follow division of the sciatic nerve in animals, and sugar has recently been detected in the urine in certain cases of sciatica (Braun).

## (IV.)—NEUROSES OF THE HYPOGASTRIC PLEXUS (NEURALGIA HYPOGASTRICA).

§ 342. Hypogastric neuralgia was first observed by Romberg, and consists, according to that observer, of painful sensations in the lower abdominal and sacral regions radiating to the upper part of the thigh and the parts supplied by the spinal hæmorrhoidal nerves. It is usually met with in females in connection with hysteria and irregularities of menstruation, and frequently occurs at the commencement of puberty. Various forms of hypogastric neuralgia may be observed according to the branches affected. In one case the utero-vaginal plexus may be affected, giving rise to hystericalgia, in another the hæmorrhoidal plexus may be the seat of the disease constituting neuralgia of the anus. At another time the ovary is the

seat of the disease, and then it is called *ovarialgia*. When the spermatic plexus is affected, it constitutes what has been described as irritable testis and neuralgia testis. At other times the urethra in men is affected, constituting *neuralgia urethralis*.

#### § 343. *Neuralgia of the Rectum.*

Neuralgia of the rectum in its pure form is rare, and there can be little doubt that a great many of the cases which have been described as such were instances of slight fissure of the anus. Dr. Anstie observed a case where the most careful examination failed to detect a fissure.

The symptoms consisted of severe paroxysms, of cutting pains coming on suddenly and spontaneously after exposure to cold, situated one inch within the anus, and greatly increased by defecation.

*Treatment.*—Besides the usual treatment of neuralgia, suppositories of morphia or subcutaneous injection of atropine (Anstie) should be used. The galvanic current may be employed locally by a suitable electrode.

#### § 344. *Uterine Neuralgia.*

The following are the causes which Dr. Anstie thought would give rise to uterine neuralgia: *Ascarides* in the rectum, profuse and intractable leucorrhœa, calculus in the kidney or ureter, prolapse of the uterus, uterine tumours of all kinds, ulceration of the cervix, and scybala impacted in the rectum, or the source of the irritation may be in some distant part of the body. In the disease which was first described by Gooch as irritable uterus, severe uterine pain, which is liable to periodical exacerbations, may exist in the absence of any recognisable disease of the pelvic organs, and the patient has generally suffered on previous occasions from neuralgia in some other part of the body.

The patient complains of paroxysmal attacks of intense pain deep in the pelvis, which is aggravated by movements, by the maintenance of the erect posture, and by pressure on the cervix. The pain often radiates to the inguinal and lumbar regions, generally on one side, and is often worse at the menstrual periods, although this is not an invariable rule.

*Vaginismus* is a condition of intense sensibility of the vaginal orifice, which renders coitus impossible. It is often associated with spasm of the constrictor vaginae and of the levator ani, and attempts at coition may induce general hysterical convulsions.

#### § 345. *Ovarialgia—Ovarian Hyperæsthesia.*

Ovarian neuralgia may be caused by any of the sources of irritation already enumerated as the causes of uterine neuralgia. It is frequently associated with grave hysterical phenomena, but it is difficult to determine whether the ovarian pain is to be regarded as the cause of the



hysterical symptoms, or as the effect of the central affection which underlies hysteria. The left ovary is most usually affected.

*Symptoms.*—Particular attention has recently been drawn to ovarialgia by Charcot and his pupils. The symptoms consist of pain, which is sometimes so acute that the patient cannot tolerate the slightest touch, and which is localised partly in the hypogastrium and partly in the iliac fossa. In many cases the pain is only discovered by deep pressure over either ovary, and in these cases there is more or less complete anæsthesia of the abdominal walls, and the seat of the pain is much more circumscribed than in the former variety. Deep pressure over the iliac region enables the investigator to feel the concave curve formed by the superior inlet of the pelvis, and towards its middle the hand will feel an ovoid body elongated transversely, and which is frequently swollen to the size of an olive or a small egg (Charcot). Pressure exaggerates this fixed iliac pain, and causes it to irradiate towards the epigastrium and throat. The irradiation of the pain towards the epigastrium is often accompanied by nausea and vomiting, and if the pressure be continued palpitations of the heart, increased frequency of the pulse, and a sensation of "globus hystericus" supervene. Besides these symptoms, which constitute the usual premonitory symptoms of a hysterical attack, or the hysterical aura, Charcot describes other symptoms in continuation of this. These symptoms are, according to Charcot, in case of compression of the left ovary, "intense sibilant sounds in the left ear, which the patients compare to the hissing noise produced by the whistle of a railway engine; a sensation as of blows from a hammer falling on the left temporal region; and, lastly, a marked obscurity of sight in the left eye." Similar phenomena occur on the corresponding parts on the right side when pressure is applied over a right hyperæsthetic ovary.

#### § 346. *Neuralgia of the Testis.*

Neuralgia of the testis is by no means so common an affection as neuralgia of the ovary. Severe neuralgic pains in the testis occur as the result of morbid growth in the organ, and as a symptom of a renal calculus descending the ureter. Dr. Anstie observed it as a reflex effect of herpes, and it is not uncommon in neurotic subjects, as the result of self abuse.

Neuralgia of the testicle consists of paroxysms of severe pain arising spontaneously and usually unilaterally, and seated partly in the testicle and partly in the epididymus and cord. The organ is tender to pressure and contact, and between the paroxysms of pain a certain amount of dull aching pain is complained of. During the attacks of pain the testicle is strongly retracted owing to spasm of the cremaster muscle, and they are frequently attended with fainting and vomiting.

This affection is usually very obstinate, and it is often accompanied by great mental depression, which may end in confirmed hypochondriasis or melancholia.

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